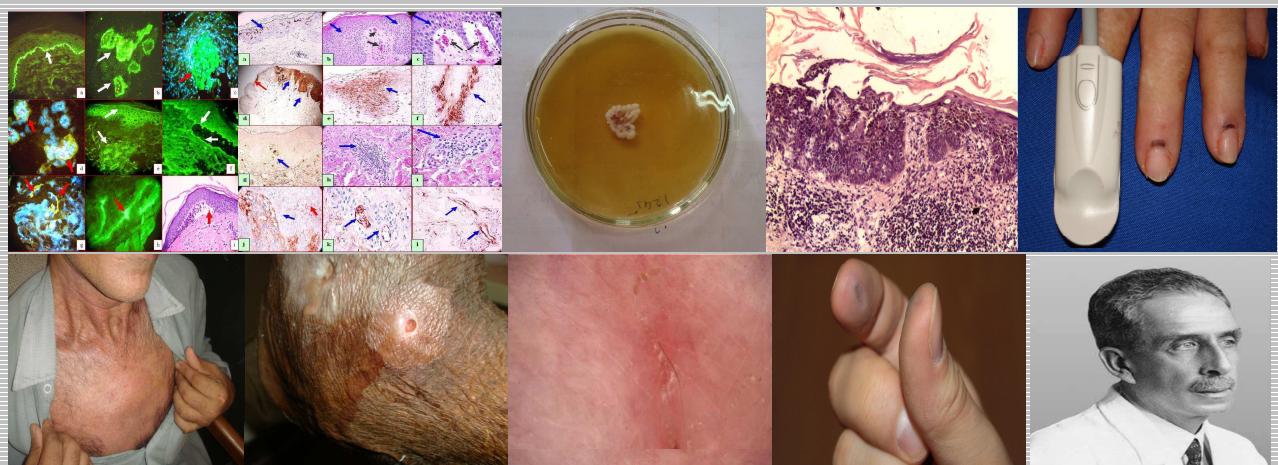


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IgG BULLOUS PEMPHIGOID WITH ANTIBODIES TO IgD, DERMAL BLOOD VESSELS, ECCRINE GLANDS AND THE ENDOMYSIUM OF MONKEY ESOPHAGUS

IgG PEMFIGOID PECHERRZOWY Z PRZECIWCIĄŁAMI IgD W OBREBIE NACZYŃ KRWIONOŚNYCH SKÓRY, GRUCZOŁÓW EKRYNOWYCH I W ENDOMYSIUM PRZEŁYKU MAŁPY

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Abstract

Context: Bullous pemphigoid is mediated by autoantibodies primarily targeting two structural proteins of basement membrane hemidesmosomes, BP180 (BPAG2; collagen XVII) and BP230 (BPAG1). **Case Report:** A 70-year-old Caucasian male patient was evaluated for a seven day history of multiple itching, erythematous blisters on his extremities. Biopsies for hematoxylin and eosin examination, direct immunofluorescence and indirect immunofluorescence (including salt split skin analysis) were performed. **Results:** Hematoxylin and eosin examination demonstrated a subepidermal blister. Within the blister lumen, numerous eosinophils and lymphocytes were noted. Direct and indirect immunofluorescence revealed linear deposits of IgG, Complement/C3 and fibrinogen at the basement membrane zone of the skin and surrounding selected dermal blood vessels and sweat glands. Positive intracytoplasmic staining for anti-human IgD was noted in most of the epidermis, as well as surrounding some dermal blood vessels. Indirect immunofluorescence utilizing monkey esophagus substrate demonstrated strong positivity within the endomysium for IgG antibodies. **Conclusion:** We report a unique case of bullous pemphigoid with reactivity to eccrine sweat glands, and selected dermal blood vessels. In addition, the observed reactivity of anti-human IgD, and of IgG to monkey esophagus endomysium warrant further investigation.

Streszczenie

Kontekst: W pemphigoidzie pęcherzowym występują autoprzeciwciała skierowane przede wszystkim przeciwko dwóm strukturalnym białkom podstawnej błony hemidesmosomalnej, BP180 (BPAG2; kolagenu XVII) i BP230 (BPAG1). **Opis przypadku:** U 70-letniego mężczyzny rasy kaukaskiej oceniono siedmiodniową historię zróżnicowanego świądu i rumieniowych pęcherzy zlokalizowanych na jego kończynach. Wykonano biopsję skóry z hematoksyliną i eozyną, immunofluorescencję bezpośrednią i pośrednią (w tym analiza splitu skóry w soli). **Wyniki:** Badania w hematoksylinie i eozynie wykazały podnaskórkowe pęcherze. W świetle pęcherzy obserwowano liczne eozynofile i limfocyty. Bezpośrednia i pośrednia immunofluorescencja wykazały liniowe depozyty IgG, komplement/C3 i fibrynogen w zonie błony podstawnej skóry oraz w wybranych naczyniach krwionośnych skóry i gruczołów potowych. Pozytywne wewnętrzcytoplazmatyczne barwienie dla anty-ludzkiej IgD odnotowano w większości naskórka, jak i otoczeniu niektórych naczyń krwionośnych skóry. Pośrednia immunofluorescencja z wykorzystaniem substratu przełyku małpy demonstrowała się silnie dodatnio w endomysium dla przeciwciał IgG. **Wnioski:** Prezentujemy unikalny przypadek pemfigoidu z reaktywnością ekrynowych gruczołów potowych i wybranych naczyń krwionośnych skóry. Ponadto obserwowano reaktywność anty-ludzkiej IgD i IgG w stosunku do endomysium przełyku małpy co skłania nas do prowadzenia dalszych badań.

Key words: bullous pemphigoid, eccrine glands, IgD, blood vessels, autoantibodies.

Słowa kluczowe: pemphigoid pęcherzowy, gruczoły ekrynowe, IgD, naczynia krwionośne, autoprzeciwciała

Introduction

Bullous pemphigoid (BP) one of the most prevalent autoimmune blistering disorders, classically presents in senior patients [1,2]. BP is a rare, persistent skin condition that usually manifests as fluid-filled blisters (bullae) on the skin. Linear pattern

autoantibodies are classically detected at the basement membrane zone (predominately IgG and Complement/C3, targeting the type XVII collagen component of the hemidesmosomes) [1,2]. Clinically, the earliest lesions may appear urticarial. Tense bullae eventually erupt, most commonly at the inner thighs and

upper arms; however, the trunk and extremities are frequently involved [1,2]. The diagnosis must be confirmed by routine histologic analysis and direct immunofluorescence (DIF) of lesional and perilesional skin, respectively [1-3]. Indirect immunofluorescence (IIF), with the use of a sodium chloride-split normal human skin substrate, further distinguishes BP from epidermolysis bullosa acquisita (EBA), which may otherwise be indistinguishable clinically, histologically, and via immunofluorescence [1-3]. Recently, multiple enzyme-linked immunosorbent assay (ELISA) tests of high sensitivity and specificity have been developed and utilized for the confirmation of BP autoantibodies.

Case Report

A 70 -year-old Caucasian male patient was evaluated for a 7 day history of several itching, erythematous blisters on his extremities. Biopsies for hematoxylin and eosin (H & E) examination, direct immunofluorescence and indirect immunofluorescence (including salt split skin analysis on monkey esophagus substrate) were performed as previously described in detail [3-7]. The patient reported no clinical symptoms of celiac disease. The patient serum also tested positive for bullous pemphigoid (BP) utilizing a commercial enzyme linked immunosorbent assay (ELISA) Mesacup test (MBL International, Woburn, Massachusetts, USA).

Results

In **Figure 1**, we highlight our most significant H&E, DIF and IIF results, including deposition analysis of basement membrane zone (BMZ) autoantibodies utilizing a 0.1M sodium chloride IIF split skin technique on normal human skin substrate. In addition, IIF findings are described utilizing monkey esophagus substrate studies. In brief, microscopic examination of the H&E tissue sections demonstrated a subepidermal blistering disorder. Within the blister lumen, numerous eosinophils are present, with occasional lymphocytes and neutrophils also seen. Within the dermis, a mild, superficial, perivascular infiltrate of lymphocytes, histiocytes and eosinophils was identified. DIF studies were performed, and displayed the following results: IgG (+++, Linear BMZ; and surrounding dermal eccrine sweat glands and selected deep dermal blood vessels); IgG3(-); IgG4(-); IgA(-); IgM(-); IgD (++, intra-cytoplasmic in epidermal keratinocytes, and surrounding selected dermal blood vessels); IgE (-); Complement/C1q(-); Complement/C3(++, linear shaggy BMZ and also within eccrine glands); albumin (++, linear BMZ and within eccrine glands) and fibrinogen (++, deep dermal perivascular). Indirect immunofluorescence (IIF) studies were performed on normal human skin substrate, and displayed the following results: IgG(++, linear BMZ, within eccrine glands, and surrounding deep dermal blood vessels); IgA(-); IgM(-); IgD(++, intra-cytoplasmic on epidermal keratinocytes, and also surrounding superficial dermal blood vessels); IgE (-); albumin (++, linear BMZ), Complement/C1q(-); Complement/C3(+++, linear BMZ); fibrinogen (++, deep dermal perivascular) and with salt split skin (++, IgG and C3 accentuated on the blister roof). Utilizing monkey esophagus and patient serum, we detected strong

reactivities at the BMZ, and also within the endomysium (fig.1).

Discussion:

Bullous pemphigoid (BP) is an autoimmune blistering disease. The most common disease associated autoantibodies are directed against two protein components of the BMZ hemidesmosomes; specifically, BP180 and BP230 [1-3]. One group of authors has investigated whether BP patients also have autoantibodies targeting plectin, another hemidesmosome protein component with extensive homology to BP230 [8]. They tested sera from 16 patients with BP, utilizing immunoprecipitation (IP) studies followed by immunoblotting (IB). Serum of one of the 16 patients with BP (6%) contained autoantibodies binding to plectin, while no reactivity was found with sera from three control subjects. Sera from all 16 BP patients immunoprecipitated BP230 from extracts of biosynthetically radiolabelled human keratinocytes [8]. The authors concluded that sera from BP patients might contain autoantibodies binding to plectin. Notably, bullous pemphigoid antigen 1, plectin, desmoplakin, envoplakin, and periplakin are all members of the plakin protein family of cytolinkers. These proteins are present within eccrine glands and the dermal/epidermal BMZ [9].

The BMZs of the skin and its appendices are very complex biochemical structures, including other molecules such as alpha integrin 2, which has been described in developing eccrine glands [10]. In addition, it has been shown that human eccrine glands recapitulate human epidermal structures and constituents. Proteins such as filaggrin, loricrin, involucrin, envoplakin, periplakin, and transglutaminases I and III match the constitutional pattern of normal human skin, and the structural qualities of junctional complexes and hemidesmosomes are preserved. [11]. Both the eccrine gland and dermal/epidermal BMZs classically contain collagen IV CIV) [12]. Fibronectin and laminin are also conserved molecules within epidermal and eccrine gland hemidesmosomes [13-15]. Further, monkey esophagus endomysium contains CIV. Thus, we suggest that BP patients may develop autoantibodies to any of these proteins, expressed in multiple anatomic structures. Further studies are necessary to further confirm this possibility, and to determine potential new directions in clinical treatment.

Acknowledgement

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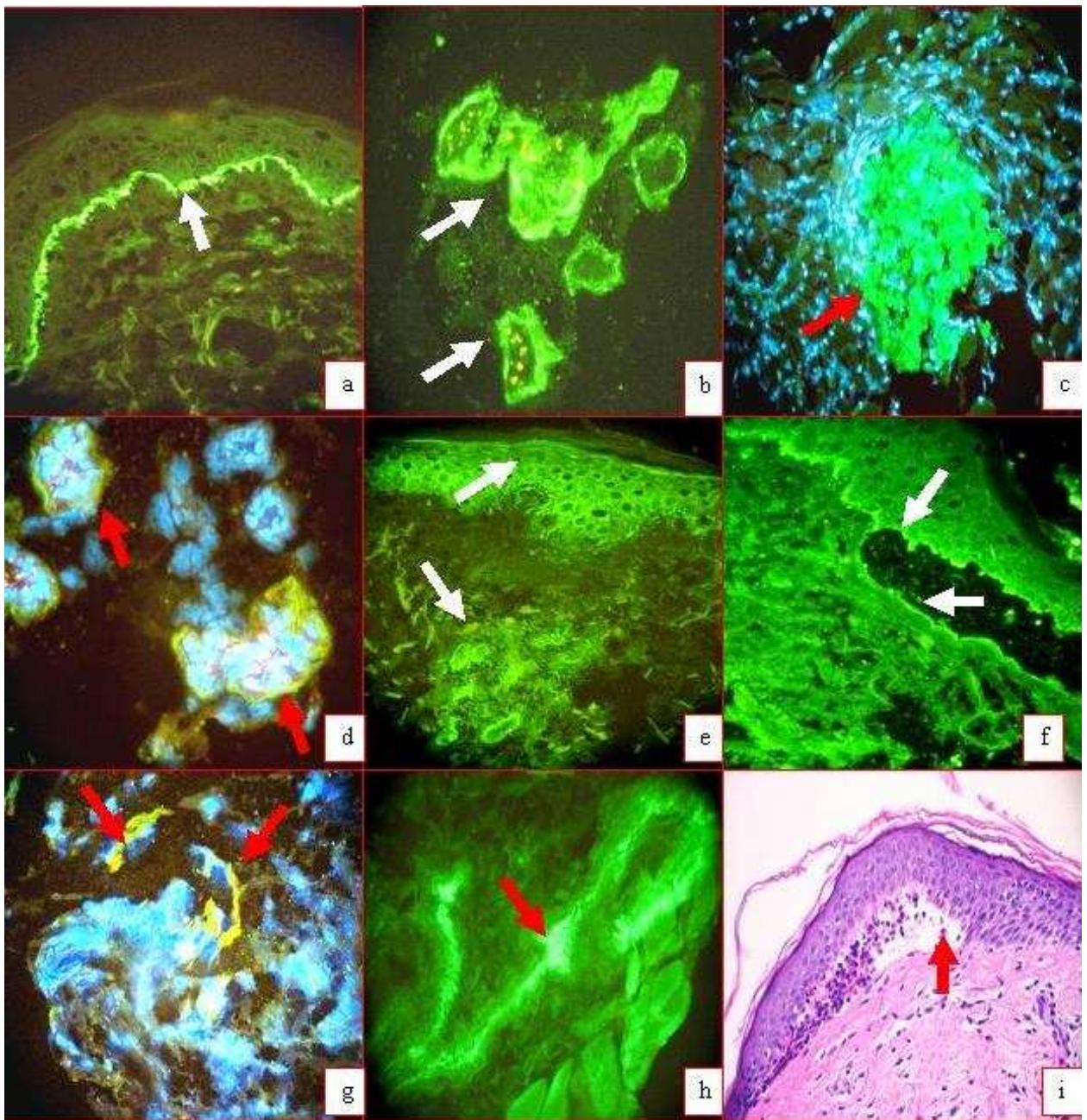


Figure 1. **a**, FITCI conjugated Complement/C3 positive in a linear, shaggy pattern at the BMZ; and in **b**, within dermal eccrine glands (green staining, white arrows). **c** and **d**, FITCI conjugated IgG positivity. In **c**, on monkey esophagus substrate, demonstrating strong positivity to anti-endomysial antibodies; in **d**, at the periphery of dermal eccrine glands (green staining, red arrows). Nuclei are counterstained with Dapi(light blue). **e**, FITCI conjugated IgD positive intracytoplasmic staining, surrounding epidermal keratinocyte nuclei, and also noted surrounding dermal blood vessels (green staining, white arrows). **f**, One molar sodium chloride (NaCl) salt split skin IIF demonstrates deposits of FITCI conjugated IgG on blister roof and floor, accentuated on the roof (green staining, white arrows). **g**, Shows deposits of FITCI conjugated IgG around deep dermal blood vessels (green staining, white arrows). Selected cell nuclei are again counterstained with Dapi. **h**, Again utilizing monkey esophagus, note the strong reactivity to dermal blood vessels using FITC conjugated anti-human IgG (green-yellow staining, red arrow). **i**, H&E of a subepidermal blister, displaying a brisk infiltrate of eosinophils and some neutrophils (red arrow).

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SPONGIOTIC DERMATITIS WITH A MIXED INFLAMMATORY INFILTRATE OF LYMPHOCYTES, ANTIGEN PRESENTING CELLS, IMMUNOGLOBULINS AND COMPLEMENT

ZAPALENIE SKÓRY ZE SPONGIOZĄ, Z MIESZANYM LIMFOCYTOWYM NACIEKIEM ZAPALNYM, KOMÓRKAMI PREZENTUJĄCYMI ANTYGEN IMMUNOGLOBULINAMI I KOMPONENTEM

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Abstract

Background: The clinical and histological presentation of spongiotic dermatitis and its inflammatory infiltrates warrant further investigation. In this case documentation of a patient with cutaneous spongiotic reactivity, we aim to characterize antigen presenting cells, as well as the skin-specific cutaneous lymphocyte antigen population by multiple techniques. **Case report:** A 30 year old Caucasian female presented with a two week history of blistering and erosions around the vaginal, rectal and axillary areas. **Material and Methods:** We utilized hematoxylin and eosin histology, direct immunofluorescence, immunohistochemistry and confocal microscopy methods to evaluate the immune reaction patterns of the cutaneous inflammatory cells. **Results:** In the primary histologic areas of spongiotic dermatitis, a mixed population of B and T lymphocytes was seen. Ki-67 antigen proliferative index staining was accentuated in these areas, correlating with the presence of large numbers of epidermal and dermal antigen presenting cells. Among the antigen presenting cell population, we detected strong positivities with CD1a, Factor XIIIa, myeloid/hystoid antigen, S100, HAM-56, and CD68. Interestingly, immunoglobulins G, D and M and Complement factors C1q and C3 were also strongly expressed in antigen presenting cell areas, including positivity within the spongiotic epidermis and around dermal vessels. **Conclusions:** We document a heterogeneous population of B and T lymphocytes and the presence of multiple classes of antigen presenting cells, immunoglobulins and complement in and surrounding histologically spongiotic areas; these findings further correlated with increased levels of expression of Ki-67.

Streszczenie

Wstęp: Kliniczna i histologiczna prezentacja spongiozy w skórze i naciekach zapalnych wymaga dalszych badań. W tej dokumentacji przypadku pacjenta z reaktywną skórną spongiozą, staramy się scharakteryzować komórki prezentujące抗原, a także specyficzne dla skóry antygeny skórnego populacji limfocytów, poprzez wykorzystanie wielu technik. **Opis przypadku:** U 30-letniej kobiety rasy kaukaskiej oceniano dwutygodniową historię pęcherzy i nadżerek zlokalizowanych na całym obszarze pochwy, odbytu i okolicy pachwin. **Materiał i Metody:** Wykorzystano obrazy histologiczne z hematoksyliną i eozyną, immunofluorescencję bezpośrednią, badanie immunohistochemiczne i mikroskopię konfokalną jako skale oceny immunologicznych wzorców reakcji skórnego komórek zapalnych. **Wyniki:** W głównych histologicznych obszarach spongiozy, zapalenia skóry, obserwowano mieszaną populację limfocytów B i T. Ki-67 – antygenowy wskaźnik proliferacji barwienia korelował w naskórku i skórze z obecnością dużej liczby komórek prezentujących抗原. Wśród populacji komórek prezentujących抗原, wykryliśmy silnie pozytywną reakcję z CD1a, czynnikiem XIII, antygenem myeloid/hystoid, S100, HAM-56 i CD68. Co ciekawe, immunoglobuliny G, D i M oraz dopełniacz C1q i C3 były równie mocno wyrażone w obszarach komórek prezentujących抗原, w tym pozytywnie w obszarach spongiozy naskórka i skóry oraz wokół naczyń skórnego. **Wnioski:** Udokumentowaliśmy heterogeniczną populację limfocytów B i T oraz obecność wielu klas komórek prezentujących抗原, immunoglobuliny i komponent w histologicznych obszarach spongiozy; te wyniki dalej korelowły ze wzrostem poziomu ekspresji Ki-67.

Key words: antigen presenting cells, B and T lymphocytes, endothelial cells, Ki-67, complement, immunoglobulins

Słowa klucze: komórki prezentujące抗原, limfocyty B i T, komórki endotelialne, Ki-67, komplement, immunoglobuliny

Abbreviations and acronyms: Confocal microscopy (CFM), immunohistochemistry (IHC), direct immunofluorescence (DIF), hematoxylin and eosin (H&E), antigen presenting cells (APCs).

Introduction:

Eczema is a common skin condition, histologically manifested as a spongiotic dermatitis. Patients experience intense pruritis that, if not controlled, can lead to secondary excoriation, with resultant infection and scarring. The first manifestation of this condition may occur at young ages. Eczema predilects male patients at all ages [1-3]. Classically, it presents on the abdomen, chest or buttocks. Head and scalp presentations are unusual. A hereditary component has been postulated to contribute to the disease etiopathogenesis. An individual affected with eczema experiences itching or pain; these symptoms are accompanied by cutaneous inflammation, clinically manifested as the classic skin rash [1-3]. The rash may also develop fluid-filled blisters [1-3]. Other clinical causes of spongiotic dermatitis include allergic contact or systemic reactions to foods, plants, metals, dyes and medications. Infants may develop spongiotic dermatitis via an allergic contact diaper rash.

Other clinical causes of a spongiotic dermatitis include environmental irritants, perfumes, smoke, and solvents; stress, hormone fluctuations, exposure to UVA/UVB solar radiation (especially if the patient is photosensitive), and climate changes [1-3]. Spongiotic dermatitis may initially be identified by its characteristic erythematous rash. If untreated, the condition may progress and become chronic; the rash may darken in color and become rough and crusty. Exacerbated spongiotic dermatitis may display vesicles (small blisters filled with fluid) or bumpy, erythematous skin with pronounced pruritis. Topical medications are thus used to reduce both itching and inflammation. If the symptoms can be controlled, the disease progression will often be halted; thus, the possibility of permanent scarring is minimized [1-3].

If presenting pruritis is not accompanied by a significant rash, then a menthol-based cream or lotion may be utilized [1-3]. If the pruritis is thus not controlled, or if severe symptoms exist, then a topical corticosteroid may be prescribed. The topical corticosteroid will address both pruritis and inflammation. If topical treatments are ineffective, a prescription for an oral corticosteroid such as prednisone may be given to the patient. Some patients also report that taking Vitamin A or fish oil has provided relief from symptoms. Keeping the affected area moist with any kind of non-irritating lotion or cream is useful in reducing irritation. In our patient, a topical moisturizer and topical corticosteroid were prescribed with improvement of her lesions [1-3].

Case report:

A 30 year old Caucasian female presented in consultation to the dermatologist with two week history of blistering and erosions in the vaginal, rectal and axillary areas. The clinical history was relevant for childhood allergies. No previous adult history of allergies to food, deodorants, sanitary towels or cosmetics was noted. The clinical examination demonstrated vesicles and erosions in erythematous, edematous areas. The history and clinical examinations for sexually transmitted diseases were negative.

Methods:

We performed two lesional skin biopsies from clinical blisters. The first biopsy was fixed in 10% buffered formalin, and submitted for hematoxylin and eosin (H&E) and periodic acid Schiff (PAS) examination, as well as for immunohistochemistry (IHC). The second biopsy was placed in Michel's transport medium, and submitted for direct immunofluorescence (DIF). The H&E, IHC and DIF studies and stains were performed as previously described [4-8].

Results:

Examination of the H&E tissue sections demonstrated diffuse, florid epidermal spongiosis. Serum scale crust was present, and intraepidermal Langerhans cell microabscesses were noted. Early evidence of a subepidermal blistering disorder was seen, although frank blister formation was not observed. The dermis displayed a florid, superficial and deep, perivascular and interstitial infiltrate of lymphocytes, histiocytes, eosinophils, neutrophils and mast cells. Plasma cells were rare. No definitive evidence of an infectious, or a neoplastic process was observed. Focal, dermal perivascular leukocytoclastic debris was noted, but frank vasculitis was not appreciated (fig. 1,2,3).

Direct immunofluorescence (DIF): was performed, and evaluated via the following grading system: (+) weak positive to (++++) strong positive, and (-) negative. Results of the DIF were IgG (+, Intracytoplasmic epidermal keratinocytic, and dermal perivascular); IgG4(-); IgA(-); IgM(-); IgD(-); IgE (-); Complement/C1q(++, Focal BMZ and superficial dermal perivascular); Complement/C3(++, Focal BMZ and superficial dermal perivascular); albumin (+++, diffuse, nonspecific dermal) and fibrinogen (+++, focal epidermal and florid, diffuse dermal) (fig 2,3).

Confocal microscopy (CFM): We performed CFM examinations utilizing standard 20X and 40X objective lenses; each photoframe included an area of approximately 440 x 330 μm . Images were obtained using EZ-1 image analysis software (Nikon, Japan).

Discussion:

Spongiotic dermatitis (SD) is often encountered in routine dermatology and dermatopathology practice. Spongiosis is a term used to describe the dermatopathologic appearance of an epidermis impacted by intercellular edema. Resultant spaces are present between epidermal keratinocytes, which may progress to intraepidermal vesiculation. The pathophysiologic mechanism of spongiosis remains unknown. It has been proposed that keratinocyte apoptosis induced by T-cells affects transmembrane proteins involved in cell to cell adhesion; the protein alterations may then be responsible for development of spongiosis via dermal hydrostatic pressure [1-3]. Additional histologic features of spongiotic dermatides include serum crust, lymphocytic exocytosis, and Langerhans cell microabscesses within the epidermis.

Prior studies have attempted to characterize the inflammatory infiltrates present in spongiotic dermatitis [9-10]. A predominant T lymphocyte population has been reported [9-10]. In this study, our findings were in agreement with several authors because we also detected a large population of T lymphocytes. However, we also found several CD1a, factor XIIIa, HAM-56 and CD68 positive cells. Consistent with our findings, other authors have also reported that immunoglobulin D may play a pathophysiologic role [11]. Interestingly, we found robust B and T lymphocyte activity, and a prominent antigen presenting cell (APC) population.

Further, the reactive cell and cytokine combination has not been previously highlighted in spongiotic dermatides. The APCs participate in the initiation of the inflammatory process in various immune-mediated dermatoses, via activation of antigen specific T lymphocytes. The skin contains several different subsets of APCs. Non-professional APCs do not constitutively express the MHC class II proteins required for interaction with naive T cells; these are expressed only upon stimulation of the non-professional APC by certain cytokines such as gamma interferon (IFN- γ).

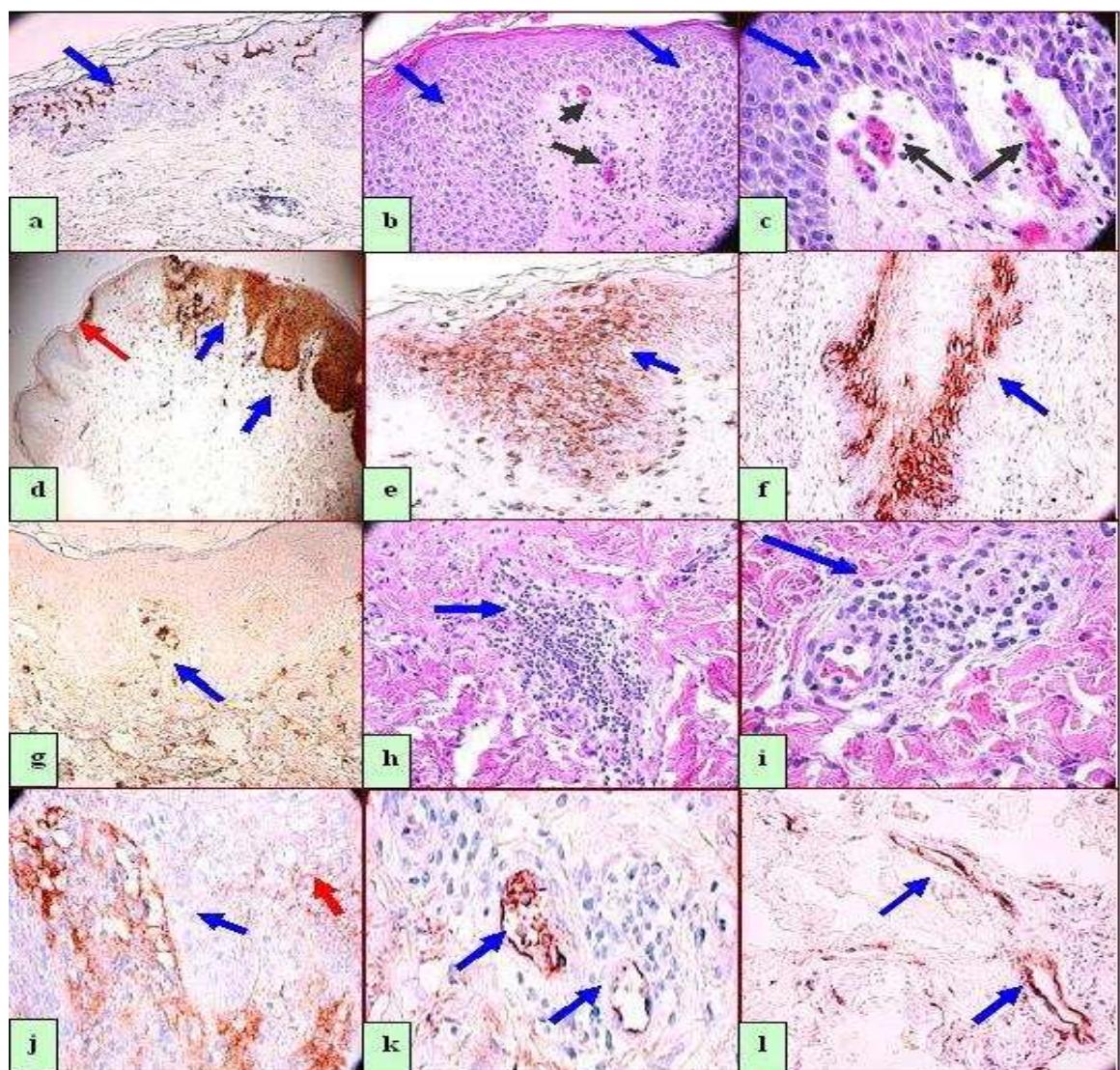


Figure 1. a. CD1a positive cells in the epidermis by IHC (brown staining, blue arrow). b. H&E sections highlighting epidermal spongiosis, with widening of the spaces between keratinocytes (100X) (blue arrows). The black arrows highlight dermal papillary tip vascular microthrombi. c. Same as b, at higher magnification (400X). d. IHC showing strong positivity to myeloid/histoid antigen in the area of spongiotic epidermis (brown staining; blue arrows), in contradistinction to markedly less staining at the specimen periphery, unaffected by the spongiosis (100X) (red arrow). e. Similar to d, at higher magnification (400X). Blue arrow indicates positive epidermal myeloid/histoid staining; red arrow indicates punctate staining in a papillary dermal tip. f. Positive Complement/C3 IHC staining around a hair follicle periphery below the spongiosis (brown staining, blue arrow). g. HAM-56 positive IHC straining around papillary dermal tip vessels where the microthrombi in b and c were seen (brown staining, blue arrow). h and i. H & E staining shows inflammation around dermal blood vessels at lower and higher magnifications, respectively (blue arrows). j. Positive IHC staining for Complement/C3 in the papillary dermis (blue arrow) and at the basement membrane zone of the skin (blue arrow); also, note fine, punctate deposits within the epidermis (red arrow). k and l. IgD positive IHC staining in deep papillary dermal blood vessels (brown staining, blue arrows).

Non-professional APCs in the skin include dermal fibrohistiocytic cells and vascular endothelial cells. In our patient, we detected multiple professional and non-professional APCs, as well as broad B and T activated lymphocytic populations in relevant areas. Indeed, the myeloid/histiocyte antigen (reactive with human cytoplasmic L1 antigen, or calprotectin) was very reactive in the zone of epidermal spongiosis, as well as in the dermis in proximity to this process [13,14].

The fact that we found complement as well as immunoglobulins in the inflamed area indicates that the immune response in an spongiotic dermatitis may be more complex than currently thought.

Thus, we recommend future studies to further investigate these findings.

In our case the primary clinical cause of the spongiotic dermatitis was not determined with certainty. The patient was treated with topical steroids and oral antihistamines, with subsequent complete improvement of her dermatosis.

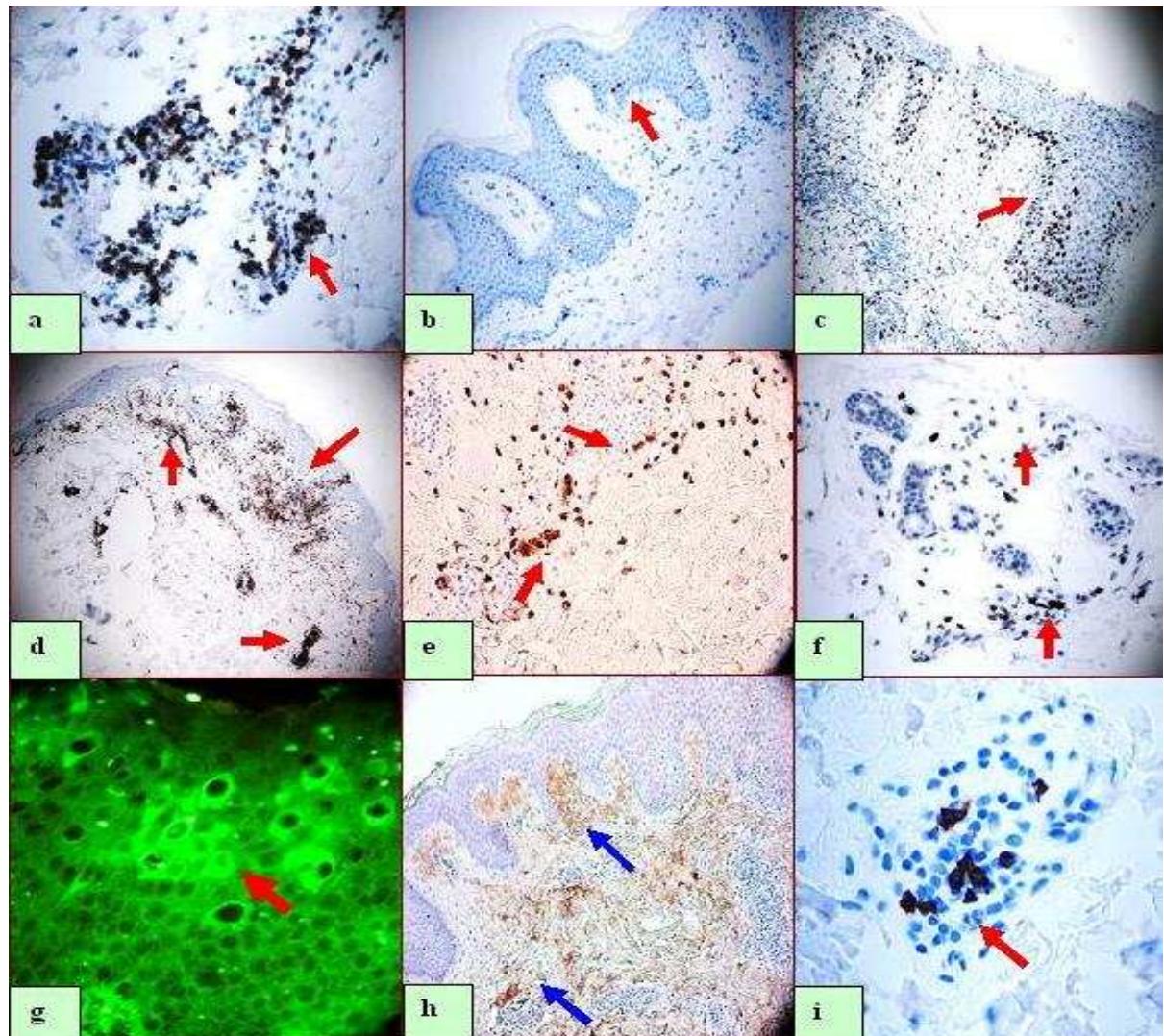


Figure 2 **a**, Epidermal CD3 IHC positive cells (dark brown staining, red arrow). **b.** and **c.** Positive Ki-67 IHC staining, in **b** on the non-spongotic edge of the skin biopsy (minimal brown staining, red arrow); in **c**, note the markedly increased staining in the histologically spongotic area (dark brown staining, red arrow). **d.** CD45 positive IHC staining, accentuated in superficial and deep dermal areas cells subjacent to the spongiosis (brown staining, red arrows) (40X). The CD45 positive staining included both CD3 and CD20 positive lymphocytes. **e.** Positive CD68 IHC staining on individual cells, grouped in the papillary dermis subjacent to the spongiosis (brown staining, red arrows). **f.** CD3 positive IHC staining on lymphocytes infiltrating eccrine glands subjacent to the spongiosis (brown staining, red arrows). **g.** DIF demonstrating positive staining with anti-human FITC conjugated IgG, in perinuclear and cytoplasmic patterns in spongiotic epidermal keratinocytes (green staining, red arrow). **h.** Positive Complement/C1Q IHC staining, diffuse in the papillary dermis in surrounding papillary dermal tip blood vessels adjacent to the spongiosis (brown staining, blue arrows). **i.** Positive CD8 IHC staining around the dermal papillary tip blood vessels (dark brown staining, red arrow).

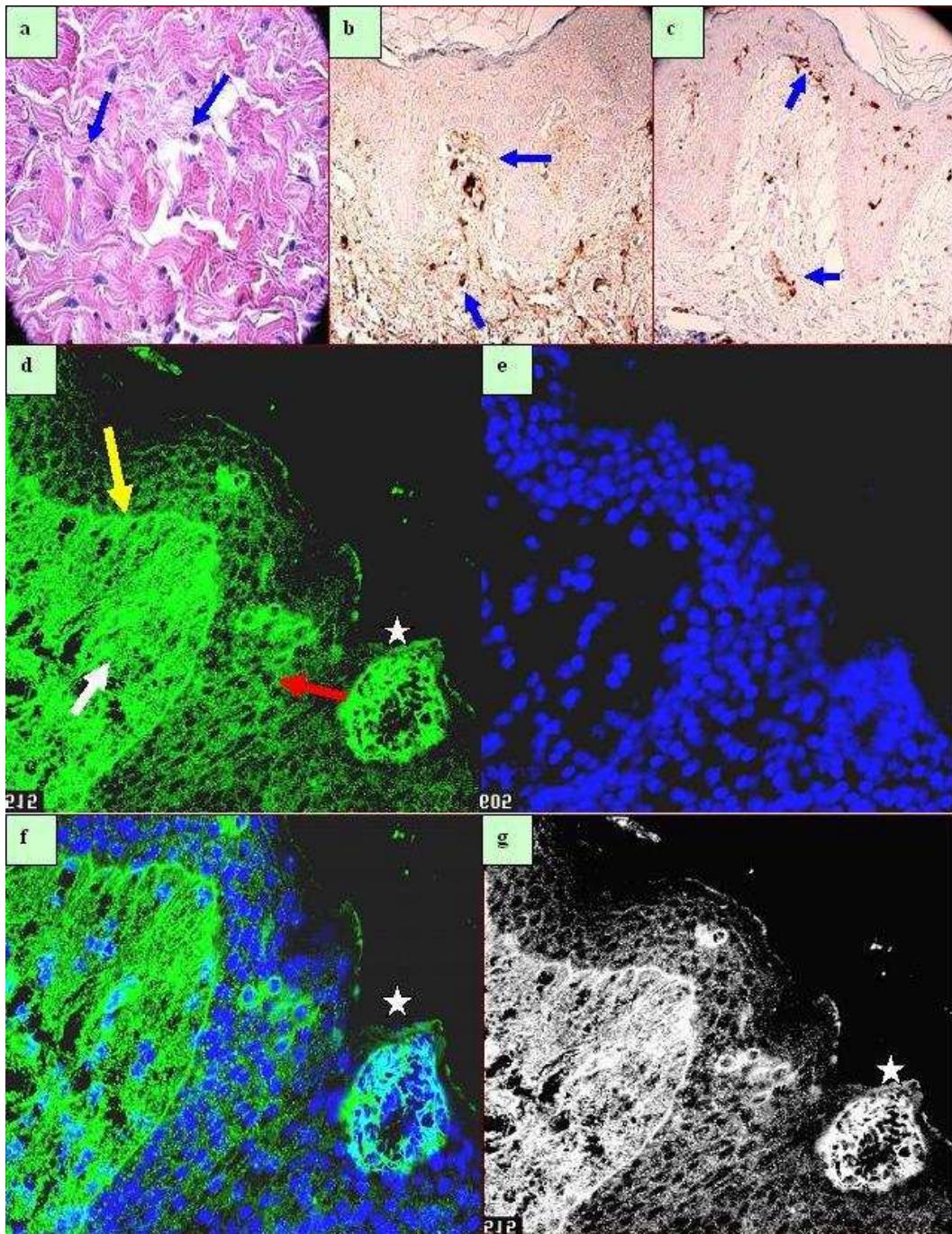


Figure 3. **a.** H & E, highlighting fibrohistiocytic cells in the dermis (grey cells, blue arrows). **b** Many of the fibrohistiocytic cells were positive for Factor XIIIa (blue arrows). **c.** Positive S100 IHC staining on epidermal and dermal Langerhans cells (blue arrows). **d** through **g** Confocal microscopy(CFM). In **d** and **f**, staining for FITC conjugated anti-human fibrinogen, showing in **d** shaggy linear staining at the basement membrane zone of the dermal/epidermal junction (yellow arrow). The white stars highlight positive net-like staining, likely representing fibrinogen deposition within epidermal Langerhans microabscesses. The red arrow highlights scattered fibrinogen positive cells in the epidermis. The white arrow shows a strong papillary dermal positivity, subjacent to the primary epidermal spongiotic area. **e.** Shows positive epidermal nuclear counterstaining with Dapi (dark blue). **f**, Combined CFM staining of fibrinogen (green staining) and Dapi(blue staining). **g**, similar to **f** in black and white relief, highlighting positive fibrinogen staining on epidermal cells and cell junctions.

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PENICILLIUM MARNEFFEI - AIDS DEFINING ILLNESS PENICILLIUM MARNEFFEI – AIDS, DEFINICJA CHOROBY

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Abstract

Background: Penicillium marneffei infection is the emerging fungal infection in the present day global scenario of HIV pandemic. P. marneffei is a dimorphic fungi with mycelial growth at 37°C. Suspicion of P.marneffei infection arises when a immunocompromised individuals especially HIV positive persons present with Molluscum contagiosum like skin lesions. But pulmonary manifestations are not characteristic of P.marneffei infection unless we test the sputum for fungal growth in individuals with low CD₄ counts ,we may miss P.marneffei respiratory infection. **Material and methods:** 100 sputum samples from HIV patients with cough were examined for fungal pathogens by inoculating the samples on SDA and incubated at 28°C. The samples with greenish yellow mycelial growth with diffusible red pigment were inoculated on blood agar and SDA and incubates at 37°C for conversion to yeast. **Results:** We isolated two cases of P.marneffei out of 100 samples. The CD₄ counts of the cases were 33 and 84. **Conclusions:** Early diagnosis and treatment reduces the mortality P.marneffei HIV patients.

Streszczenie

Wstęp: Infekcja Penicillium marneffei jest pojawiającym się zakażeniem grzybiczym w obecnym, globalnym scenariuszu pandemii HIV. P. marneffei jest dymorficznym grzybem ze wzrostem grzybni w 37°C. Podejrzenie zakażenia P.marneffei powstaje, gdy u osób z obniżoną odpornością, zwłaszcza HIV stwierdzamy obecność mięczaka zakaźnego, jako zmiany skórnej. Płucne objawy nie są charakterystyczne dla infekcji P.marneffei chyba, że test plwociny jest dodatni (dla wzrostu grzybów) u pacjentów z niskim mianem CD4; możemy przeoczyć wówczas infekcję P.marneffei w układzie oddechowym. **Materiał i Metody:** Zbadano 100 próbek plwociny od pacjentów zakażonych HIV z towarzyszącym kaszlem; zbadano patogeny grzybicze poprzez zaszczepienie próbek na SDA i inkubację w temperaturze 28°C. Próbki z żółto-zielonkawym wzrostem grzybni z dyfuzyjnym, czerwonym barwnikiem zaszczepiono na krew, agar oraz SDA i inkubowano w temperaturze 37°C do konwersji na drożdżach. **Wyniki:** Stwierdziliśmy dwa przypadki izolowanych P. marneffei ze 100 próbek. Miano komórek CD4 w analizowanych przypadkach wynosiło 33 i 84. **Wnioski:** Wczesne rozpoznanie i leczenie zmniejsza śmiertelność z powodu infekcji P. marneffei u pacjentów z HIV.

Key words: infection, dimorphic, fungi, low CD₄ counts

Słowa kluczowe: zakażenia, dymorfizm, grzyby, niskie CD₄ miano

Introduction

Penicillium marneffei is known to be endemic in S E Asia. It causes infections of RE system in humans in immunocompetent & more often in immunocompromised individuals especially in AIDS patients. As a result of recent increase of HIV infection P.marneffei has become one of the principal new emerging fungal pathogens. First human infection was reported in 1959 and caused by accidental puncture of finger by a needle used to inoculate hamsters in Segretain who had given the name P. marneffei. First spontaneous infection in humans was reported in 1973 in a splenic abscess case. Second case was reported in 1984 as a focal pulmonary infection. During the period of 1988-89 disseminated P. marneffei infection began to be observed in AIDS patients [1] and it is also included as

an AIDS defining illness among patients who have lived or visited endemic areas. At present it is considered to be the third most frequent opportunistic pathogen after tuberculosis and cryptococcosis in endemic areas [2].

Material and methods

Two consecutive sputum samples at an interval of 3 days were collected from HIV positive patients, whose CD₄ cell counts are less than 500 /_{cumm}, as shown in Table no. 1, with complaint of cough and fever for more than one week, in a sterile wide mouthed container. Patients were asked to wash their oral cavity with distilled water before collecting sputum in order to avoid contamination of sputum with commensal flora from oral cavity.

Sputum was inoculated on two sets of Sabouraud's dextrose agar (SDA with antibiotic gentamicin alone and SDA with gentamicin and cycloheximide) and incubated at 25°C +or - 2°C in BOD for 4 weeks. SDA bottles were examined for growth once in two days during 1st week and twice a week thereafter up to 4 weeks.

SDA medium with growth was processed by standard methods. LPCB mount was done for filamentous growth. Growth was identified by arrangement of conidia. Slide cultures were done to demonstrate hyphal and conidial arrangement. When two samples yielded the same fungal isolates, then only they were considered as pathogenic.

CD ₄ Count	Males	Females
< 100	6	0
101-200	13	2
201-300	15	13
301-400	20	7
401-500	13	11
	67	33

Table 1. Showing CD₄ counts

Results

P. marneffei is a dimorphic fungi. At 25°C on SDA grows as a mycelial fungus producing rapidly growing greenish yellow sporulating colony with a red centre and dark green edges with diffusible brick red pigment (Fig.1). At 37°C on SDA it produces smooth glabrous off white yeast like growth with little pigment (fig. 2). Microscopically the fruiting heads sometimes have terminal conidia larger than the ones beneath them called Corda's phenomenon, characteristic of *P. marneffei* (fig. 3).



Fig. 1 Growth of *P.marneffei* on SDA at 25°C with diffusible red pigment



Fig. 2 Growth of *P.marneffei* on SDA at 37°C
glabrous off white yeast like growth



Fig. 3 Photomicrograph of *P.marneffei* showing
Corda's phenomenon

Discussion

Clinical picture includes fever, lymphadenopathy, hepatosplenomegaly, leucocytosis, anaemia, persistent cough, molluscum contagiosum like lesions and disseminated infection. Pulmonary manifestations like cough, dyspnoea, occasionally chest pain .haemoptysis associated with pneumonia, pulmonary abscess or pulmonary infiltrates are seen. We isolated two cases of *P.marneffei* from HIV positive individuals with cough of more than one week duration who attended the ART centre, KGH,Visakhapatnam. The CD₄ counts of the two individuals are 33 and 84 respectively. We got permission from Local Ethics Committee, Andhra Medical College, Visakhapatnam to conduct the study Annexure-I. *Penicillium marneffei* was isolated for the first time in and around Visakhapatnam. Bhagyabati Devi S. et al. isolated *P.marneffei* from sputum of HIV positive individuals

whose CD₄ counts were less than 100 (21.4% positivity) in Imphal [3]. *P. marneffei* is a potentially fatal disease in the absence of treatment as documented by a case fatality rate of 91.3% in immunocompetent individuals and 100% in AIDS patients. As Penicilliosis is highly susceptible to Iatrakonazole, it can be used in the treatment as well as in secondary prophylaxis and also in primary prophylaxis [1].

Conclusions

So early diagnosis and timely treatment reduces the mortality from *P. marneffei*.

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ENFERMEDAD DE BOWEN TRATADA CON CRIOTERAPIA COMBINADA CON IMIQUIMOD TOPICO AL 5%. TRATAMIENTO ALTERNATIVO A LA CIRUGÍA EN PACIENTES MAYORES CON CO-MORBILIDADES BOWEN'S DISEASE TREATED WITH CRYOTHERAPY COMBINED WITH TOPICAL 5% IMIQUIMOD. ALTERNATIVE TREATMENT TO SURGERY IN ELDERLY PATIENTS WITH CO-MORBIDITIES

CHOROBA BOWENA LECZONA KRIOTERAPIĄ POŁĄCZONĄ Z MIEJSCOWYM 5% IMIQUIMODEM. ALTERNatywne leczenie w stosunku do operacji u osób starszych z chorobami współistniejącymi

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Resumen

La enfermedad de Bowen (EB) es un carcinoma epidermoide in situ en el cual existen cambios displásicos en todo el espesor de la epidermis. Afecta generalmente a personas de piel clara de más de 60 años. Clínicamente se caracteriza por pápulas y placas solitarias o múltiples, eritematodescamativas, de crecimiento centrífugo lento. El diagnóstico diferencial de la EB debe establecerse con dermatosis crónicas como la psoriasis, el ecema crónico, el carcinoma basocelular superficial y la enfermedad de Paget cutánea. Solo un 5% de casos progresa hacia carcinoma epidermoide invasor. Presentamos el caso de una mujer de 63 años de edad con EB tratada con crioterapia combinada con imiquimod tópico al 5% que respondió satisfactoriamente a esta combinación terapéutica.

Summary

Bowen's disease (BD) is an in situ squamous cell carcinoma in which dysplastic changes occur throughout the full thickness of the epidermis. It usually affects fair-skinned people over 60 years. It is characterized by erythematous papules and plaques solitary or multiple, with a slow centrifugal growth. The differential diagnosis of BD should be established with chronic dermatoses such as psoriasis, chronic eczema, superficial basal cell carcinoma and Paget's disease of the skin. Only 5% of cases progress to invasive squamous cell carcinoma. We report the case of a woman of 63 years of age with BD treated with cryotherapy combined with topical 5% imiquimod who responded adequately to this combination therapy.

Streszczenie

Choroba Bowena (BD) jest rakiem kolczystokomórkowym in situ, w którym zachodzą zmiany dysplastyczne na całej grubości naskórka. Zwykle dotyczy osób o jasnej karnacji w wieku powyżej 60 lat. Charakteryzuje się rumieniowymi grudkami i tarczkami, pojedynczymi lub mnogimi, z powolnym wzrostem odśrodkowym. Diagnostykę różnicową BD należy przeprowadzić z przewlekłymi chorobami skóry takimi jak łuszczyca, wyprysk przewlekły, powierzchowny rak podstawnokomórkowy i choroba Pageta skóry. Tylko 5% przypadków przechodzi w inwazyjnego raka kolczystokomórkowego. Opisujemy przypadek kobiety w wieku 63 lat z BD lezoną krioterapią, połączoną z miejscowym 5% imiquimodem, która odpowiedziała adekwatnie na tą skojarzoną terapię.

Palabras clave: enfermedad de Bowen, carcinoma in situ, crioterapia, Imiquimod.

Key words: Bowen's disease, in situ carcinoma, cryotherapy, Imiquimod.

Słowa kluczowe: choroba Bowena, rak in situ, krioterapia, Imiquimod

Introducción

El carcinoma epidermoide in situ, también llamado enfermedad de Bowen, descrito por John T. Bowen en 1912, afecta principalmente a adultos, mayores de 60 años en el 80% de los casos y es ligeramente más frecuente en mujeres [1]. Las lesiones se pueden presentar tanto en piel cubierta (75%) como en piel expuesta al sol (25%), principalmente en cabeza, cuello y tronco, aunque en población negra se ha encontrado con mayor frecuencia en extremidades inferiores [2].

La decisión terapéutica a tomar en cada paciente dependerá de la experiencia de cada profesional y de las condiciones clínicas de cada paciente ya que no existe ningún protocolo estandarizado.

Caso clínico

Mujer, 63 años de edad, ama de casa, procede de medio urbano, con hipertensión arterial mal controlada, que consultó por una placa eritematodescamativa de 10 años de evolución en pierna izquierda, de aparición espontánea. Fue creciendo lenta y asintomáticamente.

Al examen físico se trataba de una placa eritematosa ovalada con descamación blanquecina fina y adherente, bien delimitada, bordes regulares, de 7 x 5 cm. de diámetro, localizada en cara anterior de pierna izquierda (fig. 1).

Se le realiza una biopsia incisional que demuestra un carcinoma epidermoide in situ: enfermedad de Bowen (fig. 2).



Figura 1. Clínica de la lesión. Placa eritematosa ovalada con descamación blanquecina fina, adherente y áreas con costras amarillentas, bien delimitada, bordes regulares, de 7 x 5 cm. de diámetro, localizada en cara anterior de pierna izquierda.

Figure 1. Clinical. Erythematous oval plaque with thin flaking white, sticky and yellowish crusty areas, well-defined, regular borders, 7 x 5 cm. in diameter, located on left leg.

Se indica aplicación de imiquimod al 5% tópico, 1 vez/día/3 veces por semana por 4 semanas y luego se realiza crioterapia, bajo anestesia tópica con EMLA®. Para la aplicación de nitrógeno líquido se empleó un equipo CryAC conectado a una punta A de 3.8 cm. de diámetro. Se realiza en forma secuencial en número de

dos, por 10-15 segundos para así congelar tanto el tumor en su totalidad como el margen de piel sana de 7 mm por fuera del tumor. La temperatura final alcanzada, empleando el pirómetro y las agujas termopares, fue de -60°C con dos ciclos completos de congelación-descongelación. A los 15 días presenta una erosión con costras miéliceras y se indica antibioticoterapia oral. A la semana de inicio del antibiótico la lesión no presenta signos de impetiginización por lo que se suspende. A los 8 meses del tratamiento se presentó una cicatriz atrófica levemente eritematosa con áreas de piel normal (fig. 3). Se tomó una biopsia de control que mostró un área de fibrosis dérmica y ausencia de neoplasia intraepidérmica residual (fig. 4).

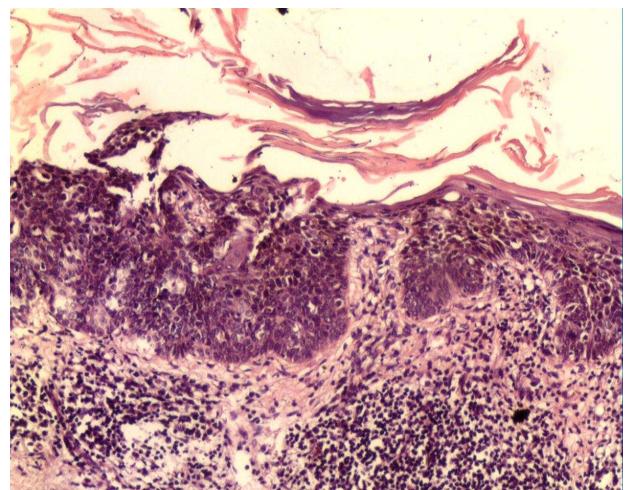


Figura 2. Histología de la lesión. Epidermis con acantosis, pérdida de polaridad y maduración celular que afecta todo el espesor epitelial. Hiperqueratosis. Denso infiltrado de mononucleares en dermis (HE 10X).

Figure 2. Histology. Epidermis with acanthosis, loss of polarity and cell maturation that affects the entire epithelial thickness. Hyperkeratosis. Dense infiltrate of mononuclear cells in dermis (HE 10X).

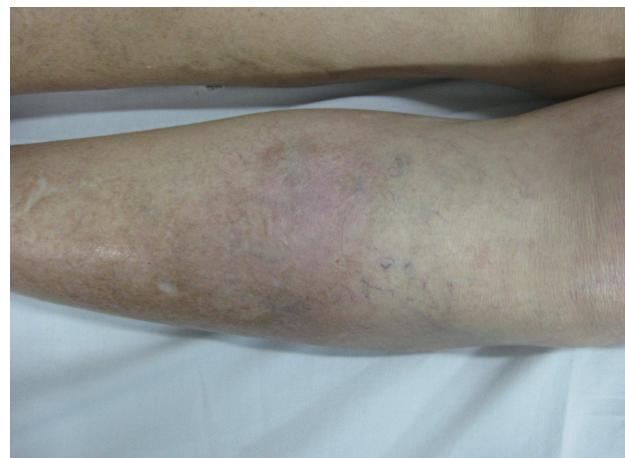


Figura 3. Clínica post-tratamiento. Cicatriz atrófica levemente eritematosa con áreas de piel normal. Figure 3. Clinical post-treatment. Slightly erythematous atrophic scar with normal skin areas.

Comentario

La EB es de etiología desconocida. Está relacionada con los siguientes factores predisponentes: exposición crónica al sol, contacto con arsénico y derivados, radioterapia y agentes virales como el virus del papiloma humano (VPH) [3-5].

En general son lesiones únicas y sólo en una quinta parte de los casos son múltiples; forman placas eritematosas con escama fina, ocasionalmente fisuradas, de tamaño variable y contorno irregular pero bien definido que tienden a extenderse gradualmente en forma anular o polícílica [6-8].

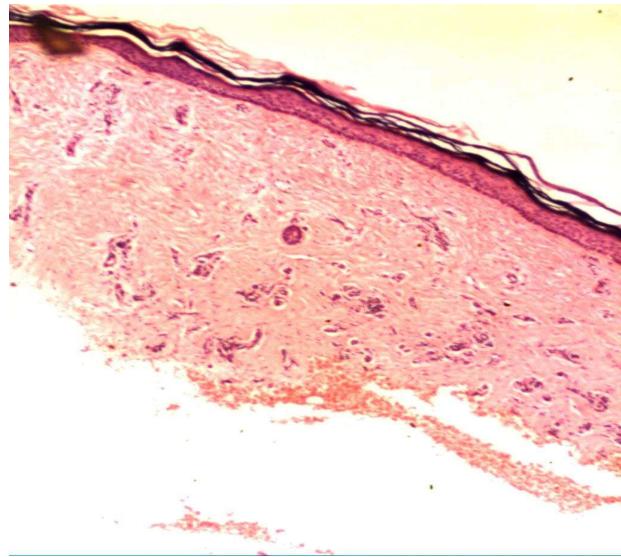


Figura 4. Histología post-tratamiento. Epidermis adelgazada con pérdida de redes de crestas. Fibrosis dérmica. Ausencia de restos neoplásicos (HE 4X).

Figure 4. Histology post-treatment.- Thinned epidermis with loss of rete ridges. Dermal fibrosis. Absence of neoplastic remnants (HE 4X).

El diagnóstico diferencial incluye a la dermatitis crónica, psoriasis, queratosis seborreica, carcinoma basocelular superficial, carcinoma metastásico, melanoma de extensión superficial en las lesiones pigmentadas y verrugas vulgares en lesiones queratósicas [1].

En la histología se observa pérdida de polaridad de los queratinocitos, atipia y mitosis en todo el espesor de la epidermis. Queratinocitos multinucleados o con núcleos hiperchromáticos y nucléolos prominentes, las células basales son normales. La dermis superficial muestra infiltrados de linfocitos, histiocitos y células plasmáticas [9].

Del 5% al 20% de los casos desarrolla carcinoma epidermoide invasor, que al menos en un tercio da metástasis. El 14% de los pacientes presenta múltiples lesiones premalignas y malignas junto con la EB [10].

El tratamiento es muy variable, siendo la cirugía convencional el tratamiento estándar, aunque algunos pacientes no pueden someterse a ella debido a la edad o estado de salud. En otras ocasiones las lesiones presentan un tamaño gigante o localizaciones especiales, por lo cual los resultados serían poco cosméticos, pudiendo llevar incluso a pérdida de la funcionalidad. En dichos casos, otras opciones terapéuticas incluyen: cirugía micrográfica de Mohs curetaje, criocirugía, láser con

CO₂, radioterapia, terapia fotodinámica, 5 fluoruacilo tópico, imiquimod tópico, inyecciones locales de interferón alfa o bleomicina [7-10]. En nuestra paciente se decidió combinar dos terapias, la criocirugía y el imiquimod al 5% tópico ya que se trataba de una mujer con hipertensión que no se encontraba controlada. El tratamiento produjo una remisión rápida del proceso neoplásico sin ninguna complicación mayor, salvo la impetiginización a nivel local que superó en días.

La criocirugía es una técnica que se basa en la aplicación de un agente criogénico (el más empleado es el nitrógeno líquido) directamente sobre la piel para provocar congelación y destrucción del tejido. Este procedimiento se ha utilizado para el tratamiento de diversas enfermedades cutáneas, tanto benignas, como premalignas y malignas [1]. Para su correcta aplicación es importante conocer el agente criogénico, la técnica y las características de la lesión, tales como tamaño, localización, profundidad, agresividad y proximidad con estructuras vasculares o nerviosas. Tiene una serie de ventajas como: realizarse en forma ambulatoria, ser económica, disminuir el riesgo de infecciones agregadas, emplear sólo anestesia local, cuidados postoperatorios mínimos y resultado estético de aceptable a excelente en la mayoría de los casos [9].

El Imiquimod es un modificador de la respuesta inmune de uso tópico que ocasiona actividad antiviral y antitumoral a través de la estimulación de la inmunidad innata y adquirida. Su aplicación se realiza tópicamente, sin vendaje oclusivo, 3 días a la semana durante 5 semanas [10]. En cuanto a su respuesta inmune actúa activando macrófagos y otras células mediante su unión a receptores de superficie, induciendo la secreción de citoquinas pro-inflamatorias del tipo interferón α, factor de necrosis tumoral α e interleuquina 2 y otras del tipo interleuquina 1α, 6, 8, 12, interferón γ [4].

Lo que si se debe tener en cuenta al elegir la terapéutica es aquella que sea económica, eficaz, estéticamente aceptable y que presente menos riesgos en un paciente mayor de 60 años y con co-morbilidades.

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HEMATOMA OF THE PROXIMAL NAIL. REPORT OF 41 CASES

KRWIAK PROKSIMALNEJ CZĘŚCI PAZNOKCIA. RAPORT Z 41 PRZYPADKÓW

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Abstract

Background: The proximal fold is an important part of the nail apparatus it contributes to the formation of the nail plate and through the cuticle acts as an impermeable barrier protecting it from any cause.

Objective: To know the proximal nail fold hematoma caused by the use of pulse oximeter.

Material and Methods: A descriptive study was conducted in 41 patients with proximal nail hematoma secondary to the use of oximetry in patients hospitalized in the Intermediate and Intensive Care Unit at the Hospital General de Enfermedades from December 1, 2007 to December 31, 2010.

Results: We studied 41 patients with proximal nail fold hematoma secondary to the use of oximeter, 30 (73.1%) were males and 11 (26.8%) females. The numbers of fingers affected by pulse oximeter were in one digit 30 (73.1%) cases, in two digits 6 (14.6%), in three digits 3 (7.3%), in 4 digits 1 (2.4%) and in 5 digits 1 (2.4%) case. The most affected proximal nail fold was right index: 24 (58.5%), right middle 11 (26.8%), right ring 6 (14.6%), left index 12 (29.2%), and left middle 6 (14.6%) cases.

Conclusions: Hematomas of the proximal nail fold may be caused by different traumas. The use of pulse oximeter is one of them.

Streszczenie

Wstęp: Proksymalny wał jest ważną częścią paznokcia, przyczynia się do powstawania płytka paznokciowej i poprzez działania oskórka jako nieprzepuszczalnej bariery chroni go.

Cel: Obserwacja krwiaków proksymalnego wału paznokcia powstały po użyciu pulsoksymetru.

Materiał i metody: Opisowe badanie zostało przeprowadzone u 41 pacjentów z wtórnym, proksymalnym krwiakiem paznokcia po zastosowaniu oksymetrii u chorych hospitalizowanych w Oddziale Intensywnej Terapii w Szpitalu General de Enfermedades od 1 grudnia 2007 do 31 grudnia 2010.

Wyniki: Zbadano 41 pacjentów z wtórnym krwiakiem proksymalnego wału paznokcia powstały po użyciu oxymeteru. 30 badanych (73,1%) stanowili mężczyźni, a 11 badanych (26,8%) kobiety. Numery palców na których założono pulsoksymetry były następujące: palec pierwszy (kciuk) 30 (73,1%) przypadków, drugi 6 (14,6%), trzeci 3 (7,3%), czwarty 1 (2,4%) i piąty 1 (2,4%) przypadek. Najczęściej zmiany dotyczyły bliższego wału paznokcia prawej strony, indeks: 24 (58,5%), prawa połowa 11 (26,8%), prawy pierścień 6 (14,6%), indeks lewej strony 12 (29,2%), a środkowa 6 (14,6%) przypadków.

Wnioski: Krwiaki bliższego wału paznokcia mogą być spowodowane przez różne czynniki traumatyczne. Użycie Pulsoksymetru jest jednym z nich.

Key words: nail fold, oxymeter, hematoma

Słowa kluczowe: wał paznokciowy, pulsoksymetr, krwiak

Introduction

The nail apparatus may be affected by different forms of hematomas, depending on the cause different structures can be affected; the nail plate is the most affected secondary to major and minor trauma in both fingernails and feet.

The function of the proximal nail fold is to protect the nail plate through the cuticle; it acts as an impermeable barrier protecting it from any noxa. We report our experience of 41 cases with proximal nail hematoma secondary to use of the oximeter in the Intermediate and Intensive Care Unit at the Hospital

Material and Methods

A descriptive study with 41 patients presenting proximal nail hematoma secondary to the use of oximetry hospitalized in the Intermediate and Intensive Care General at the Hospital General de Enfermedades from December First 2,007 to December 31, 2,010.

Results

We studied 41 patients with proximal nail fold hematoma secondary to use of oximeter (Fig. 1), 30 (73.1%) were males and 11 (26.8%) were females; age ranging from 0-20 0 cases, 21-40 6 (14.6%), 41-60: 16(39%), 61-80 13 (31.7%) and more than 80 years 6 (14.6%) cases.

The causes of hospitalization were sepsis 12 (29.2%) cases (Fig. 2), pancreatitis 4 (9.7%), diabetes mellitus and sepsis 4 (9.7%), respiratory distress syndrome 4 (9.7%), myocardial infarction 3 (7.3%), exploratory laparotomy 3 (7.3%), craniotomy 2 (4.8%), upper gastrointestinal bleeding 2 (4.8%), chronic renal failure 2, congestive heart failure, dengue hemorrhagic fever (Fig. 3), myasthenia gravis (4.8%), Guillain Barre syndrome and bradycardia, 1(2.4%) case each one. Hematoma of the proximal nail fold were diagnosed in 41 cases and 69 digits were affected, the numbers of fingers affected by pulse oximeter were in one digit 30 (73.1%) cases, in two digits 6 (14.6%) (Fig. 4), in three digits 3(7.3%), in 4 digits 1 (2.4%) and in 5 digits 1 (2.4%) case.

The fingers affected were right index: 24 (58.5%), right middle 11 (26.8%), right ring 6 (14.6%), left index 12 (29.2%), and left medium 6 cases (14.6%) (Table 1).

The time of occurrence of the hematoma ranged from less than 10 days 20 patients (48.7%), 10-20 days: 5 (12.1%), 20-30 days 1 (2.4%), unknown 15 (12.1%) cases, less time being 3 days and the most being more than 30 days.

The left and right index fingers were the most affected because they are the digits in which oximetry is often placed

Number of affected fingers	No	%
One	30	73.1
Two	06	14.6
Three	03	7.3
Four	01	2.4
Five	01	2.4
Total	41	
Affected Proximal Nail Fold	No	%
Right index finger	24	58.5
Right middle finger	11	26.8
Right ring finger	06	14.6
Left Index finger	12	29.2
Left middle finger	06	14.6

Table 1. Hematoma of the proximal nail fold Affected Fingers



Figure 1. Oximeter in a patient in Unit Intensive Care

Discussion

Proximal nail folds of fingers can be affected by traumatic factors being one of the most frequent causes of hematomas at this level, within which we have this nail biting and tearing of hangnails. However, it is important to know other

hematomas at this level as are those produced by the oximeter use; their presence in several digits and no history of oximeter use thereof must make us suspect other diseases that can be presented such as collagen diseases or sepsis.

The proximal fold is a continuation of the dorsal skin of the digits, it gives rise to two epithelial surfaces, the dorsal and the ventral, the latter contributes to the formation of the nail plate [1].

The proximal fold is structurally similar to the surrounding skin without dermatoglyphics and sweat glands, it has three parts: the glabrous skin, the cuticle and the ventral portion of this fold called eponychium. This fold is important because it contributes to the formation of the nail plate through the dorsal matrix in the low ridge of its ventral portion, influences the growth direction of the nail plate in an oblique form above the nail bed and in the microcirculation that provides useful information on some pathologic conditions [2].

This nail fold can be affected by skin, systemic and infectious diseases, benign and, malignant tumor, drug reactions, traumas between others [2-4].

Among the traumatic causes affecting this fold are the hematomas caused by the use of oximetry in patients hospitalized at the intensive care unit, this kind of hematoma is caused by the constant pressure of the oximeter, since this is like a clip [1]. This occurs in patients in critical care where pulse oximetry is one of the most important advances to monitor blood oxygen saturation noninvasively [5].

The proximal nail fold hematoma affects the free edge letting off the cuticle, at the beginning it was thought that its appearance could be due the prolonged use of the oximeter (30 days), however some cases have been seen after 1 to 3 days of using it, one or more folds can be affected according to where the oximeter is placed [1].



Figure 2. Proximal nail fold hematoma in female patient with sepsis



Figure 4. Proximal nail fold hematoma caused by oximeter in two digits



Figure 3. Proximal nail fold hematoma in male patient with Hemorrhagic Dengue

Proximal fold hematomas can also be of iatrogenic origin due to the improper use of the oximeter [5].

Proximal fold hematomas can occur without any changes of the nail apparatus but dyschromia caused by them, unlike the ones cause by major trauma that may present with onychomadesis, onycholysis, deformity or loss of the nail and sometimes onychodystrophy [6].

It is important to recognize these traumatic hematomas of the proximal fold caused by the use of the oximeter, and distinguish them from the capillaries thrombosis of the proximal fold seen in collagen-vascular diseases, especially systemic sclerosis and dermatomyositis, as well as septicemia [1].

Bleeding from the proximal folds may also occur in mountain climbers following a severe freeze or in people who practice winter sports outdoors. This bleeding may be caused secondary to the cold that affects the peripheral circulation in the capillaries around the nail folds, causing necrosis by cell destruction, tissue edema and thrombosis. However, these mechanisms are not well defined [7].

This type of hematoma disappears on its own and all that is needed to prevent its formation is to rotate the oximeter in the different fingers of the patient. It is also important to train medical and nursing staff about the correct technique for the placement of the oximeter [1,5]. This is probably an unknown condition to many for what we consider important its disclosure.

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MANUBRIOSTERNAL JOINT INVOLVEMENT IN PSORIATIC ARTHRITIS

ZAJĘCIE SPOJENIA RĘKOJEŚCI MOSTKA W ŁUSZCZYCOWYM
ZAPALENIU STAWÓW

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Abstract

Psoriatic arthritis (PsA) is a type of seronegative arthritis associated with psoriasis of skin and nails. It affects axial and peripheral joints with variable severity. The course and prognosis of the disease suggest that early diagnosis and aggressive treatment are important. The most common clinical type of PsA is oligoarthritis in which four or fewer joints are involved often in an asymmetrical pattern, and arthritis mutilans which is the destructive form and is the least common. We report a case of PsA in which the manubriosternal joint alone was involved as initial manifestation in association with psoriatic erythroderma. .

Streszczenie

Łuszczykowe zapalenie stawów (ŁZS) jest typem seronegatywnego zapalenia stawów związanym z łuszczyką skóry i paznokci. Wpływa na osiowe i obwodowe stawy w zmiennym natężeniu. Przebieg i rokowanie choroby sugerują, że wczesne rozpoznanie i agresywne leczenia są istotne. Najczęstszą kliniczną postacią ŁZS jest oligoarthritis, w którym do czterech lub mniej stawów, często asymetrycznie objęte są procesem chorobowym oraz arthritis mutilans, które jest destruktynną formą ŁZS i jest najmniej powszechnie. Przedstawiamy przypadek ŁZS, w którym zajęte było spojenie rękojeści mostka co stanowiło wstępową manifestację zmian związaną z ertrodermią łuszczykową.

Key words: Psoriasis, Psoriatic arthritis, manubriosternal joint

Słowa klucze: łuszczyca, łuszczykowe zapalenie stawów, spojenie rękojeści mostka

Introduction

Psoriatic arthritis (PsA) is a specific form of inflammatory arthritis associated with psoriasis of skin and nails and is usually seronegative for rheumatoid factor. Clinically, five different patterns of the diseases have been described: distal interphalangial arthritis ,oligoarthritis,polyarthritis,arthritis mutilans and spondyloarthropathy [1]. These clinical patterns can change with time and may develop into more destructive forms of the disease. It is therefore suggested that early diagnosis may prevent these adverse outcomes of PsA [2,3]. We hereby report a case of PsA in the form of monoarticular involvement of manubriosternal joint associated with psoriatic erythroderma.

Case Report

A 50 year old male patient who was a known case of psoriasis presented with a two weeks history of acute exacerbation of the disease. Patient was a diagnosed case of psoriasis vulgaris for twenty years and

his disease used to get controlled by topical medications but two weeks back the disease got flared up after intake of some ayurvedic drugs. It was associated with fever and malaise. There was no history suggestive of joint pains or any other systemic disease, however, the patient complained of persistent pain over the anterior chest which started three days after the aggravation of disease. On examination, the patient was in erythroderma (Fig. 1) and typical psoriatic plaques were present on scalp and dorsum of hands and feet. Nail examination showed pitting and onycholysis in some of the finger nails. Tenderness and swelling was noted over manubriosternal joint (Fig. 2) and arthritis of the joint was suspected. Rest of the joint examination was insignificant. General physical and systemic examination was normal. Routine investigations like complete blood counts, urine examination, liver function tests and kidney function tests were normal. Serum uric acid was normal (5.6mg/dl). The synovial fluid aspirated from the inflamed joint was negative for crystal deposits.



Figure 1. Showing psoriatic erythroderma



Figure 2. Showing involvement of manubriosternal joint

Rheumatoid factor and ANA were normal. Chest x-ray and ECG were normal. An MRI scan of the sternum revealed narrowing of the joint space and articular erosions confirming arthritis of the manubriosternal joint (Fig. 3). In view of the constellation of clinical, laboratory and radiological findings, a diagnosis of PsA of manubriosternal joint was made. The patient was given weekly methotrexate (25mg/wk) and the skin lesions and arthritis showed good response after two weeks and almost resolved completely after 8 weeks of treatment.

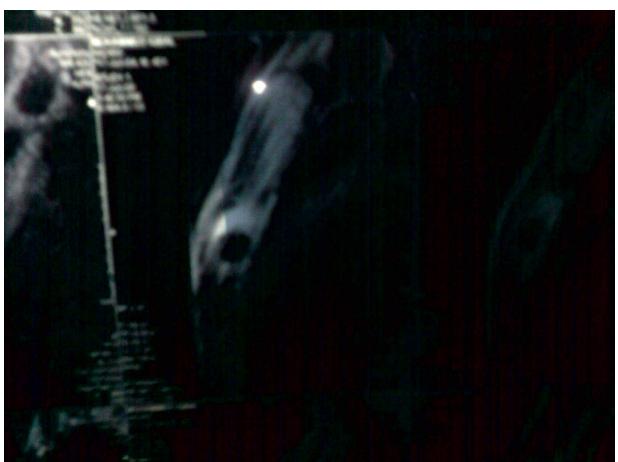


Figure 3. Radiological involvement of the manubriosternal joint on MRI

Discussion

Psoriatic arthritis (PsA) is classified as seronegative spondyloarthropathy associated with HLA-B27. The prevalence of psoriatic arthritis in general population is reported to be 0.5 percent whereas in psoriatic population it affects about 5-30 percent patients. The diseases can start at any age but the usual age of onset is between 35 and 45 years about 10 years later than psoriasis [4]. However, in 15 percent cases it can precede the diagnosis of psoriasis. The Moll and Wright classification includes five clinical groups: asymmetrical oligoarthritis, symmetrical rheumatoid like pattern, predominantly distal interphalangial type, predominantly axialarthritis which includes spondylitis and sacroilitis and arthritis mutilans which is a destructive form of PsA. Psoriasis of the nails occur in about 75 percent PsA patients as compared to 30 percent with skin lesions alone [5]. Besides the affection of axial and peripheral joints, the involvement of temporomandibular joint is not uncommon in PsA [6]. The involvement of sternal joints as initial manifestation is unusual in PsA. The association of manubriosternal joint arthritis with palmoplantar pustulosis and psoriasis vulgaris has been reported [7]. Nancy et al reported a case of manubriosternal joint arthritis in a patient of generalized pustular psoriasis without involvement of any other axial or peripheral joint [6]. In our case, the patient had psoriasis for two decades but there was no evidence of any joint involvement. The monoarticular involvement of manubriosternal joint in association with psoriatic erythroderma has not been reported earlier and to the best of our knowledge this is the first report of this association. The various treatment options in PsA are NSAIDS, intraarticular corticosteroids, systemic retinoids, immunosuppressants such as cyclosporine, azathioprine, and methotrexate, leflunamide. PUVA photochemotherapy and sulfasalazine are used successfully. Recently, TNF-alfa antagonists such as infliximab, etanercept and adalimumab are being increasingly used and are reserved for more severe cases. We treated our patient with weekly methotrexate only.

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THE MADURA FOOT - A CASE REPORT STOPA MADURSKA – OPIS PRZYPADKU

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Abstract

Madura foot or mycetoma is a chronic granulomatous disease characterized by localized infection of subcutaneous tissues by actinomycetes or fungi. The inflammatory response can extend to the underlying bone. Mycetoma was described first in the mid 1800s and was initially called Madura foot. The infection can be caused by true fungi (eumycetoma) in 40%, or filamentous bacteria (actinomycetoma) in 60%. Actinomycetoma may be due to *Actinomadura madurae*, *Actinomadura pelletieri*, *Streptomyces somaliensis*, *Nocardia* species. The infection, which may remain latent for a time, forms small, subcutaneous swellings that enlarge, soften with pus, and break through the skin surface, with concurrent invasion of deeper tissues. Sulfonamide, iodide, and antibiotic therapy have been used against actinomycotic infections, but the fungi are more resistant to treatment. We reported a patient of madura foot from International Medical College Hospital, Tongi, Gazipur. A 82-years old male was admitted to the International medical college hospital with a 16 months history of swelling with multiple discharging sinuses filled with granules localized in his right foot. Pus was examined by gram staining and periodic acid Schiff (PAS) staining. Moderate number of filamentous branching gram positive bacilli were found. The organism was recognized as a member of the *actinomyces* genus. PAS staining did not reveal any other organism. The aggressive course and progression of the disease affected the short bones of the involved foot. The patient was diagnosed as a case of Madura foot and was treated in the same hospital.

Streszczenie

Stopa Madurska lub mycetoma jest przewlekłą ziarniniakową chorobą charakteryzującą się zlokalizowanym zakażeniem tkanek podskórnych przez promieniowce i grzyby. Reakcja zapalna może rozciągać się na kości. Mycetoma po raz pierwszy została opisana w połowie 1800 roku i była początkowo nazywana stopą Madurską. Zakażenie może być spowodowane przez grzyby prawdziwe (eumycetoma) w 40% lub nitkowate bakterie (actinomycetoma) w 60%. Actinomycetoma może być spowodowana przez *Actinomadura madurae*, *Actinomadura pelletieri*, *Streptomyces somaliensis*, *Nocardia* species. Zakażenie, które może pozostawać w formie utajonej przez jakiś czas, tworzy małe, podskórne obrzęki, mogące się powiększać, pokrywać ropną wydzieliną i przebić się przez powierzchnię skóry, przy jednoczesnej inwazji w głębszej położone tkanki. Sulfonamidy, jodyna i antybiotykoterapia zostały użyte przeciwko infekcji actinomycotycznej, ale grzyby te są bardziej odporne na leczenie. Opisaliśmy pacjenta ze stopą Madurską z International Medical College Hospital, Tongi, Gazipur. 82-letni mężczyzna został przyjęty do International Medical College Hospital z 16-to miesięczną historią wielu obrzękniętych, podminowanych zatok, wypełnionych granulkami, zlokalizowanych w jego prawej nodze. Wydzielina ropna została zbadana poprzez barwienie gram oraz cykliczne barwienie kwasem Schiffa (PAS). Stwierdzono umiarkowaną liczbę rozgałęzionych, nitkowatych pałeczek gram dodatnich. Organizm został uznany za członka z rodzaju promieniowców. Barwienie PAS nie ujawniło żadnych innych organizmów. Agresywny przebieg i postęp choroby wpłynął na krótkie kości stóp. Pacjent był diagnozowany jako przypadek stopy Madurskiej i był leczony w tym samym szpitalu

Key words: Madura foot, *Actinomyces*, granules, pathogens, Bangladesh

Słowa kluczowe: Stopa madurska, *Actinomyces*, ziarnistości, patogeny, Bangladesh

Introduction

Madura foot is endemic in the tropics and subtropics. It is a deep mycosis caused by exogenous fungus or actinomycotic species. These infections lead to progressive inflammation of the skin, subcutaneous tissue, muscles and bones. The organism enters through local trauma in the foot, hand or eyes from saprophytic soil. After entry to the body they form subcutaneous nodules containing suppurative granulomas, multiple cavities and sinus tracts. The sinus tracts discharges exudates with fine grains. These grains are colonies of causal organism [1,2].

Due to the existing socio-economic condition and low living standard of the people of this area the disease is often neglected in the initial stage. Usually the diagnosis is made at an advanced stage. Good clinical response with proper pharmacological therapy alone has been reported [3]. Surgical debridement with prolonged treatment with antifungal and antibiotic has been proved effective in many cases [4]. Amputation of limbs followed by antifungal and antibiotic therapy and reconstruction have been done for a number of cases [5]. Surgical excision is necessary when bone is involved as detected radiographically [6].

In this article we are presenting a case of madura foot diagnosed and treated in a medical college hospital located at suburbs of Dhaka with limited investigation facilities. The diagnosed patient was treated with oral penicillin followed by application of local crystalline penicillin G powder with which the patient responded well. The characteristic radiological presentation, macroscopic and microscopic features are discussed in this report.

Case Report

An 82-year-old male farmer from Tongi, Gazipura presented to the International Medical College hospital on July 24, 2010 with a history of fall in a ditch in January 2009. Following that there was pain and swelling in his right foot. He was treated by local doctors with analgesic and antibiotic. The wound healed within a few days. After three months the foot again started swelling and at this time he noticed a tender, nonerythematous swelling in the same area that drained spontaneously and the wound healed. Over the next few months a number of similar smaller painless lesions were appeared in the adjacent area when the patient presented to us.

On physical examination, there was five discharging sinuses over the dorsal surface of the right foot. The sinuses were discharging pus and sulfur granules (fig.1). The patient was otherwise in good health without any sign of immunodepression. No difficulty in his walking was reported.

Laboratory investigations revealed Haemoglobin -70%, WBC total count - 8000/ cmm, ESR 48 mm in 1st hour, RBS 6.1 mmol/l , Serum creatinine-0.80 mg/dl, SGPT- 38u/l , SGOT - 24.5u/l , Urine R/E , X-Ray chest and ECG were normal. X-Ray of the right foot showed sign of destruction of the navicular bone (fig.2). Microscopic picture of fine needle aspiration showed branching filamentous bacilli (Gram positive). Some scattered pus cells are also seen (fig.3).

The patient was treated with oral penicillin followed by installation of local crystalline penicillin G powder. The patient responded well in 8 weeks of treatment and was discharged from hospital on 26th November 2010.



Figure 1. Discharging sinuses on the dorsal aspect of right foot discharging white pus filled with granules.



Figure 2. Radiograph of right foot showing destruction of the navicular bone.

Discussion

Madura foot was first recognized in 1842 by Gill in Madura district of Tamilnadu in India [7]. Later Bidie and Carter gave a full description of the disease [8,9]. Mycetomas are frequent in the tropical zones of America (Mexico and Venezuela), Africa (Senegal, Mauritania and Sudan) and Asia (India), but can also be observed in other areas. Fungi, that in these rainy areas are found as saprophytes in the soil, are usually introduced through skin wounds in those who walk bare footed (farmers, nomads) and are often exposed to penetrating wounds. Infection begins in the skin and subcutaneous tissue causing local papular or nodular swelling which tends to grow and rupture, forming communicating sinus tracts through which mucous containing the characteristic colored grains is discharged. Some sinuses heal with scarring while fresh sinuses appear elsewhere, leading to enlargement and disfigurement of the affected limb. Eventually destruction of bone occurs when grains invade the cortical margins and replace the spongiosa. General complaints are rare, and fever usually a sign of secondary bacterial infection. The infection does not, in general, spread hematogenously although cases are known where particularly *Pseudallescheria boydii* and *Nocardia asteroides* in immunocompromised patients (leukemia, HIV, use of corticosteroids and immunosuppressive drugs) have disseminated hematogenously to the brain, myocardium and the thyroid gland [10,11]. The combination of the clinical picture (indurated swelling of the foot with multiple sinuses that discharge pus filled with grains), macroscopically typical grains and the histopathological appearance is characteristic of the diagnosis. Grains vary from 0.2 to 3.0 mm in diameter and can be black, white, yellow, pink or red, depending on the microorganism involved [8,12]. On microscopy a hematoxylin-eosin (HE) stain is generally able to demonstrate and identify the characteristic grains. They are surrounded by inflammation with polymorphonuclear leukocytes, epithelioid cells, plasma cells and multinucleated giant cells with areas of fibrosis. Two groups of mycetoma are distinguished : Eumycetoma (caused by eumycetes or true fungi) and Actinomycetoma (caused by fungi like aerobic bacteria from the actinomycetes species).

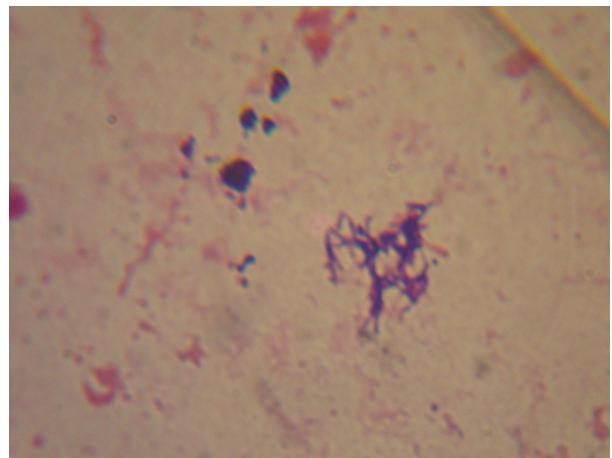


Figure 3. Microscopic picture shows branching filaments (gram positive).Scattered pus cells are also visible.

Although particular species of dermatophytes are known for their mycetoma - like infection as well, they do not lead to destruction of the bone and therefore are not considered real mycetoma [13,14]. Gram staining can be used for recognition of branching hyphae within the actinomycetes grains, while periodic acid Schiff (PAS) - staining is suitable for identification of the hyphae of eumycetes. Confirmation of the diagnosis and exact identification of the species require culture. Although theoretically more accurate than histology, culture is difficult practically and also facilities for culture was not available in this institute.

Although the clinical picture is characteristic, diagnostic confusion may occur with chronic bacterial osteomyelitis, especially when bone destruction has occurred. Botromycosis can give a similar picture; it is a chronic bacterial infection caused by gram- positive cocci (Staphylococci, Streptococci) and gram negative bacteria (Escherichia coli, Pseudomonas, Proteus) that can lead to subcutaneous swelling with draining fistulas. Like mycetoma, grains (colonized of bacteria) can be found in the suppurative discharge. In botromycosis, however, organs can be affected by the process too. Neoplasms (benign and malignant) should be excluded as well [15].

Commencement of treatment at an early stage is necessary to reduce the suffering of the patient and to prevent complication. Treatment of mycotic mycetomas is often unrewarding. It is based on surgical excision since chemotherapeutic agents (ketoconazole, itraconazole) are expensive and often not effective [16-22]. A delayed diagnosis may require extensive excision which may not always be adequate and more taxing to the patient. In our case, the actinomycetomas , patient responded well in 8 weeks with oral penicillin and local crystalline penicillin G powder. Penicillin is a very cheap and relatively safe drug without any remarkable side effect for most individuals. We advised the patient for follow up visits to detect any recurrence. For most drugs, it is recommended to continue treatment for at least 10 to 12 months and many of these drugs are toxic. As those drugs are toxic the patients need regular follow-up of hematologic, liver and kidney function parameters.

Conclusion

We reported a case of actinomycosis in this article. Although it is a rare disease, it might be encountered in our regular practicing life specially in a country where more than 75% of the people are working barefooted in the fields. Immediate and meticulous conservative and surgical measures could be of great benefit for these patients.

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DERMOSCOPY OF SCABIES DERMOSKOPIA W ŚWIERZBIE

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Abstract

We presented a female patient a 63 year-old affected with scabies and personal history of previous arthropathic psoriasis and arterial hypertension. We aimed to described clinically widespread itchy rash vs *scabies* submitted to dermoscopic examination. We confirmed diagnosis of scabies clinically and dermoscopically.

Streszczenie

Zaprezentowaliśmy 63 letnią chorą na świerzb z osobistą dodatkową historią w postaci łuszczycowego zapalenia stawów i nadciśnienia tętniczego. Naszym celem było opisanie klinicznie powszechnych, świadczących zmian w przebiegu świerzbu obserwowanych w badaniu dermatoskopowym. Potwierdziliśmy rozpoznanie świerzbu badaniem klinicznym i dermatoskopowym

Key words: scabies, dermoscopy, skin disease

Słowa klucze: świerzb, dermatoskopia, choroby skóry

Scabies is an itchy rash caused by a little mite that burrows in the skin surface. Scientific name of human scabies mites is *Sarcoptes scabiei* var *hominis*. The mite is an obligate human parasite and cannot live more than three days without a human host, but it can survive up to a month when living on a human. The life cycle of the mite lasts 30 days and is spent within the human epidermis. An affected host is infested by about 10-12 adult mites. After mating the male dies and female mite burrows into the outside layers of the skin where she lays up to 3 eggs each day for her lifetime. The period of 10 days is needed for maturation through larval and nymph stages. At the end, 10% of those eggs arise in mature mites [1].

The symptoms of the condition can last for months or even years.

Scabies is a very contagious skin condition. The mites are transferred by direct skin-to-skin contact also during sexual contact. Others, indirect contacts etc. over bedclothes, are also possible if these have been contaminated by infected persons. Symptoms will appear from two to six weeks in people who have not previously been exposed to scabies infection. In patients with previous infestations symptoms develop within one to four days after [2].

Although scabies can affect anyone of any age, race or socioeconomic group, it is more common in children and in sexually active people.

Itch is often severe. Itch tends to be in one area at first (often the hands), and then spreads to other areas. The itch tends to be worse at night and after a hot bath. A rash usually appears soon after the itch starts [3].

The rash is often most obvious on the inside of the thighs, parts of the abdomen (fig. 1) and buttocks, armpits, and around the nipples in women (fig. 2).



Figure 1. Image of clinical manifestation of scabies

Scabies is usually diagnosed by the typical symptoms and lesions and skin rash described above. Typically primary symptoms are small papules, vesicles and burrows. Secondary lesions might be various because they are the result of scratching, due to scratching attained secondary infections and sometimes outcome of the host immune response against the mites and their products.

The diagnosis can be confirmed by microscopic examination of the contents of a burrow [4].

Burrows are a pathognomonic sign and represent the intraepidermal tunnel created by the moving of female mite. Burrows are often shaped like „s“ or „z“, (fig. 3, fig. 4). Using dermoscopy we could also see the classic dermoscopic image of triangle or „delta wing jet“ sign of scabies head parts, translucent scabies body and scabies eggs (fig. 5), common seen under the breasts (as in our patients case), axillae and buttocks.



Figure 2. Clinical image of a lesion



Figure 3. Dermoscopic images of lesion



Figure 4. Dermoscopic images of lesion

A new technique, dermoscopy, makes it easier to identify the black dot at the end of a burrow which represents the mite. Examination with our Mole Max II digital dermoscope, which offers a maximum field of view of 1cm with 30-fold magnification, revealed scabies mites within minutes. The pixel resolution of each image was 640x480 at 24-bit color depth [5,6].

Our patients were treated with topical Eurax® (Crotamiton) cream/lotion which is a scabicidal and antipruritic agent. Itch disappeared almost immediately. After the treatment, skin condition was without any changes.

We conclude that dermoscopy as a accurate, non-invasive, painless, non-expensive and simple technique is important and useful tool for the diagnosis of scabies either as a diagnostic test or to guide the traditional diagnostic test [7]. Dermoscopy of scabies facilitate very low number of false-negativ results what we conceive as significant because scabies moreover may additionally mimic a host of other dermatologic conditions that can be easily misdiagnosed if not confirme.



Figure 5. Dermoscopic images of lesion

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POLYCYSTIC OVARIAN DISEASE: A DERMATOLOGIST'S VIEWPOINT

ZESPÓŁ POLICYSTYCZNYCH JAJNIKÓW: PUNKT WIDZENIA
DERMATOLOGA

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Abstract

Polycystic ovary syndrome(PCOS) is a common condition characterized by menstrual abnormalities and clinical or biochemical features of hyperandrogenism. Features of PCOS may manifest at any age, ranging from childhood (premature puberty), teenage years (hirsutism, menstrual irregularities), early adulthood and middle life (infertility, glucose intolerance) to later life (diabetes mellitus and cardiovascular disease). Cutaneous manifestations of PCOS are protean and include hirsutism, acne, androgenic alopecia and acanthosis nigricans. Treatment of cutaneous manifestations of PCOS requires a coordinated team approach. A combination of drug therapy, counseling and cosmetic procedures can maximize the results.

Streszczenie

Zespół policystycznych jajników (PCOS) jest częstym stanem charakteryzującym się zaburzeniem miesiączkowania oraz klinicznym i biochemicznym hiperandrogenizmem. Schorzenie PCOS może występować w każdym wieku, począwszy od dzieciństwa (przedwczesne pokwitanie), młodzieńczych lat (hirsutyzm, zaburzenia miesiączkowania), we wczesnej dorosłości i w połowie życia (nieplodność, nietolerancja glukozy) do późnych lat życia (cukrzyca i choroby układu krążenia). Skórnymi objawami PCOS są zmienne i to nadmierne owłosienie, trądzik, łysienie androgenowe i acanthosis nigricans. Leczenie skórnego objawów PCOS wymaga skoordynowanego podejścia zespołu. Połączenie leczenia farmakologicznego, poradnictwo i kosmetyczne zabiegi mogą zmaksymalizować wyniki.

Key words: Polycystic ovary syndrome , hyperandrogenism, hirsutism, androgenic alopecia

Słowa klucz: Zespół policystycznych jajników, hiperandrogenizm, hirsutyzm, androgennym łysieniu

Introduction

As early as 1844, Chereau described sclerocystic changes in the human ovary [1]. In 1935, Stein & Levienthal reported seven women with amenorrhea, hirsutism, obesity, polycystic ovaries.....Stein-Levienthal Syndrome [2]. Polycystic ovary syndrome (PCOS) is the most commonly encountered endocrinopathy in women of reproductive age group. It has significant reproductive and non-reproductive consequences. Women of any ethnic background can present with PCOS [3]. Several studies have suggested a prevalence of PCOS of 5-10 % in women of reproductive age group, using the diagnostic criteria proposed by the US National Institute Of Health [4]. Because patients with PCOS can present with assortment of complaints such as menstrual disturbances, infertility, hirsutism, acne, their point of entry into the medical system may be by way of a primary care physician, gynecologist, endocrinologist, or a

dermatologist. Thus all the disciplines need to be familiar with this syndrome & its long term consequences.

Definition & Diagnosis:

Historically, there has been a lack of consensus regarding the features that define PCOS. A meeting convened by the National Institute Of Health (NIH) in 1990 stressed three key features necessary for the diagnosis of PCOS [5]:

- Ovulatory dysfunction (oligo-ovulation or anovulation).
- Clinical hyperandrogenism or biochemical hyperandrogenemia.
- Exclusion of congenital adrenal hyperplasia (CAH), androgen secreting tumors, hyperprolactinemia or thyroid diseases.

Because 16-20% of normal population has polycystic-appearing ovaries on ultrasound, the presence of

polycystic ovaries was considered to be suggested but not diagnostic of PCOS [6].

Rotterdam Criteria (2 out of 3):

- Menstrual irregularity due to anovulation or oligo-ovulation.
- Evidence of clinical or biochemical hyperandrogenism.
- Polycystic ovaries (12 or more follicles in each ovary, measuring 2-9 mm in diameter and/or increased ovarian volume).

Pathogenesis:

The pathogenesis of PCOS is poorly understood, but the primary defect may be insulin resistance leading to hyperinsulinemia [7,8]. In the ovaries, the cardinal feature is functional hyperandrogenism. Circulating concentrations of insulin & LH are generally raised. The theca cells, which envelop the follicle and produce androgens for conversion in the ovary to oestrogens are over-responsive to this stimulation. They increase in size and produce androgens. This combination of raised levels of androgens, estrogens, insulin and LH explains the classic PCOS presentation of hirsutism, anovulation, dysfunctional bleeding, and dysfunction of glucose metabolism.

Signs and symptoms:

PCOS symptoms have a gradual onset. Although the symptoms can exist at the time of menarche, most of the patients do not seek help until their early mid 20's.

- Menstrual irregularities and reproductive issues:- Abnormal vaginal bleeding is a typical complaint that ranges from amenorrhea to menorrhagia & metrorrhagia. Because these patients are anovulatory, they all present with infertility issues and have an increased incidence of pregnancy loss and pregnancy associated complication [9,10].
- Obesity and metabolic abnormalities:- Prevalence of obesity is high in patients with PCOS. The rate of obesity in the PCOS population ranges from 38-87% [11]. Because obesity is associated with insulin resistance, many women with PCOS have insulin resistance, but insulin resistance in PCOS is also independent of obesity.
- Metabolic syndrome:- PCOS patients are at a higher risk for the metabolic syndrome that includes dyslipidemias, type 2 DM, hypertension and obesity [12].

The cutaneous manifestations of PCOS vary depending on the ethnic background and include these are symptoms [13]:

- Hirsutism- 66%
- Acne- 35%
- Androgenic alopecia- 6%
- Acanthosis nigricans- 3%

Hirsutism is defined as excessive facial and/or body terminal hairs in a male pattern distribution. It results from an interaction between androgens and the sensitivity of hair follicle to androgens. It occurs at puberty in response to increasing levels of androgens. Under the influence of androgens, vellus hair develops into terminal hair. Ferriman – Gallwey scoring system is used to quantify the extent of hair growth at androgen sensitive sites [14]. A score of >8 is abnormal for adult Caucasian females. Limitations of this scoring system are that it is subjective and is effected by any previous or on going treatment.

Androgenic alopecia. There is progressive loss of terminal scalp hair in genetically susceptible women with diffuse thinning of hair diameter, length and density (hairs/cm). The pattern may embrace progressive thinning of the crown with preservation of hair-line or take on a male-pattern form with bitemporal recession. Various grading systems used for grading androgenic alopecia are Ludwig's scale (grade1 – grade3) and Olsen's scale [15,16].

Seborrhea and acne are also the results of increased androgen production and/or increased skin sensitivity to androgen [17].

Acne in PCOS has following important characters:

- Persistent.
- Refractory.
- Late onset.

Associated with irregular menstrual cycles, hirsutism, obesity and androgenic alopecia.

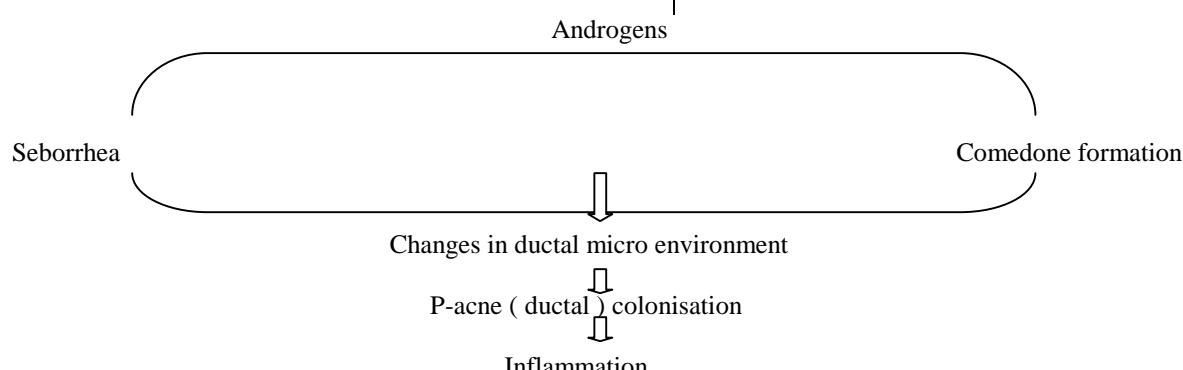


Figure 1.

Acanthosis Nigricans is characterized by hyperpigmentation and thickening of skin with papillomatous elevations. These velvety plaques are distributed bilaterally symmetrically in the neck, axillae, groins, antecubital and popliteal fossae, umbilicus and perianal areas. The exact mechanism of development of the skin lesions of acanthosis nigricans is not known, but may result from keratinocyte and dermal fibroblast proliferation stimulated by insulin and insulin like growth factors. Thus it is a Cutaneous marker of insulin resistance [18].

Treatment of hirsutism

- Direct hair removal: shaving, plucking, threading, waxing, epilators.
- Electrical depilation: it includes galvanic depilation and diathermic depilation.
- Lasers: Laser light (694-1064 nm) passes through the skin surface and is absorbed by melanin (chromophore), converted to heat energy which destroys the hair follicle. The largest is the stem cell population where pigmented cells are populated. Patients with fair skin are ideal for this procedure because darker skin carries the risk of epidermal damage as it requires higher energy pulses leading to pigmentary changes and scarring [19].
- Eflornithine 11.5% cream: It inhibits the enzyme ornithine decarboxylase responsible for catalyzing ornithine to polyamine critical to regulation of cell growth and differentiation. It slows down hair growth and reduces hair visibility and coarse nature of hair.
- Antiandrogens: Antiandrogens interfere with androgen action at the target organ either by blocking enzyme reactions or by blocking the androgen receptors [20]. A male fetus in utero is at risk of developing feminization if his mother is having treatment with an antiandrogen. So, concurrent use of adequate contraception is an essential component of any treatment regimen using an antiandrogen. Antiandrogens mostly used in hirsutism are cyproterone acetate, spironolactone flutamide, bicalutamide.
- Combined oral contraceptive pills: These suppress ovarian androgen production and thus should be the treatment of choice for mild hirsutism of PCOS [21]. But some of the progestogens in oral contraceptives have androgenic properties. It is therefore of utmost importance to choose a combination that does not have any androgenic activity. Besides being useful in hirsutism, these also cause a marked improvement in acne and seborrhea as well as a good control of menstrual cycle.
- Finasteride: It is a competitive 5-alpha-reductase inhibitor and blocks the conversion of testosterone to more potent dihydrotestosterone [22]. Comparative randomized trials have shown that finasteride (5 mg daily) has a clinical effect on hirsutism similar to that of spironolactone and flutamide. It might be also useful in women with androgenic alopecia [23].
- GnRH agonists: GnRH agonists such as Nafarelin, Buserelin, Leuprorelin decrease ovarian steroid production by suppressing LH and FSH secretion. Over a period of six months therapy, hair growth reduces in majority of patients and there is a marked reduction of seborrhea also [24].

• Ketoconazole: Its principle inhibitory role involves inhibition of the 17, 20-desmolase and 11-beta-hydroxylase steps in steroidogenesis. Because of its significant ovarian suppressive effects, its use has also been suggested for ovarian androgen suppression in hirsutism [25]. Improvement has been noted with 400mg per day in PCOS.

Other treatment options for hirsutism

- Metformin: Metformin (500mg thrice a day) is being increasingly used in PCOS. It significantly reduces hyperinsulinemia and hyperandrogenism. It causes a significant reduction in serum androgens, improvement in menstrual irregularities and resumption of ovulation, but only a mild improvement in hirsutism [26].
- Weight reduction: It is well recognized that obesity worsens hirsutism. Failure to respond to antiandrogen therapy is much more common in obese than in slim patients. Weight reduction decreases hyperinsulinemia, insulin resistance and hyperandrogenism and has thus beneficial effects on menstrual abnormalities and hirsutism [27]. Therefore low-energy diet and exercise should be encouraged as a form of first line therapy.
- Psychotherapy: It is very important to address the sociopsychological aspects of this disorder in some women. Hirsute women have increased levels of anxiety and depression. So, psychotherapy in the form of group therapy is very beneficial.

Treatment of Androgenic alopecia

- Minoxidil: Used as 2% and 5% topical solution, it increases duration of anagen and enlarges miniaturized and suboptimal follicle. About 1 ml is applied to scalp twice daily for a minimum period of four months. About 5% cases improve over a period of 48 weeks.
- Hormonal therapy: Antiandrogens and Finasteride may also be beneficial in severe cases [28].

Treatment of Acne [29-31]

- Topical Retinoids: Topical retinoids including Tretinoin, Adapalene and Isotretinoin normalizes desquamation and also decreases the inflammatory response.
- Benzoyl peroxide: It kills the microorganisms and prevents the development of severe acne scarring.
- Antibiotics: Antibiotics such as tetracycline, doxycycline, minocycline, erythromycin, azithromycin are the mainstay of therapy for acne.
- Oral Isotretinoin: It reduces sebum production, normalizes desquamation and decreases the inflammatory response.
- Hormonal therapy: In severe unresponsive cases, antiandrogens or OCP's may be administered either alone or in combination with topical or systemic antibiotics.

Treatment of Acanthosis nigricans

Treatment is only symptomatic and consists of topical application of mild keratolytics like salicylic acid ointment or retinoic acid cream. Oral Isotretinoin has also been used in some cases [32].

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TWO COLORED FINGERS SIGN (COLORED FINGERS SIGN) OBJAW DWÓCH KOLOROWYCH PALCÓW (OBJAW KOLOROWYCH PALCÓW)

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Skin lesions occur in illustrators, (draughtsman) draftsman (fig. 1-4). Skin lesions are primarily on two fingers (finger I - thumb and finger II - pointing) of different colors. Coloration generally occurs in black, and the subject is the color of pen, pencil, which is used by a person. As a result of prolonged use of the stylus (scriber) or pencil goes to chronic fingertips color of the color of the stylus. Discoloration persists chronically, but it is reversible. Disappears when the person ceases to use the stylus to perform their work or hobbies. Lesions may be accompanied by: a depression in the spot compressions held stylus (pencil), discoloration of the distal edge of the nail plate (colour pen), damage to the distal edge of the nail plate, periodic numbness in the fingers. (Piotr Brzeziński 21.01.2011).



Figure 1

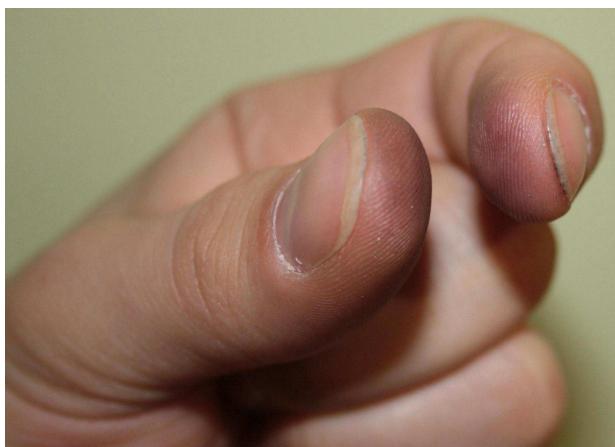


Figure 2

Zmiany skórne występują u rysowników, kreślarzy, traserów (ryc. 1-4). Zmiany skórne zlokalizowane są przede wszystkim na dwóch palcach (palec I – kciuk i palec II-wskazujący) o różnym zabarwieniu. Zabarwienie z reguły występuje w kolorze czarnym, a uwarunkowane jest kolorem rysika, ołówka którym posługuje się dana osoba. W wyniku długotrwałego posługiwania się rysikiem lub ołówkiem dochodzi do przewlekłego zabarwienia opuszek palców w kolorze rysika. Przebarwienie utrzymuje się przewlekle, ale jest odwracalne. Zanika wówczas gdy osoba zaprzestanie używać rysika do wykonywania swojej pracy lub hobby. Zmianom barwnikowym mogą towarzyszyć: nieznaczne wgłębenie w miejscu uciśnięcia przez trzymany rysik (ołówek), zmiany zabarwienia dystalnego brzegu płytki paznokciowej (w kolorze rysika), uszkodzenie dystalnego brzegu płytki paznokciowej, okresowe drętwienie palców. (Brzeziński Piotr 21.01.2011).

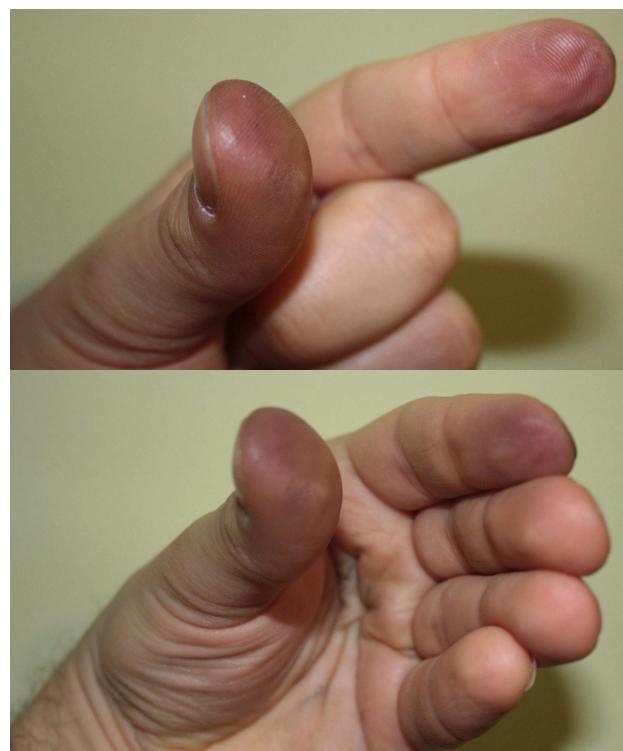


Figure 3,4

DERMATOLOGY EPONYMS – PHENOMEN / SIGN – DICTIONARY (C)

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CARBOXYHEMOGLOBIN SIGN

The bright red coloration skin and internal organs due to carbon monoxide poisoning

OBJAW KARBOKSYHEMOGLOBINOWY

Jaskrawoczerwone zabarwienie skóry i narządów wewnętrznych z powodu zatrucia tlenkiem węgla.

CARDINAL SIGNS

(of inflammation), known as dolor, calor, rubor, tumor and functio laesa. These are the signs of acute inflammation as described by A.C. Celsus about 2000 years ago. Due to the release of certain chemical mediators we get calor and dolor; the result of increased blood flow with blood vessel congestion. Dolor and tumor are the result of increased permeability of blood vessels with blood and fluids escaping outside the vessels.

OBJAWY KARDYNALNE

(zapalenie), znane jako ból, ocieplenie, zaczernienie, obrzmienie, upośledzenie funkcji. Są to objawy ostrego zapalenia opisane przez Celsusa A.C. około 2000 lat temu. Ze względu na wyzwolenie niektórych mediatorów chemicznych mamy ból i ocieplenie; wynik zwiększonego przepływu krwi z zatorów naczyń krwionośnych. Ból i obrzmienie są wynikiem zwiększonej przepuszczalności naczyń krwionośnych i ucieczka płynów poza naczynia.

AULUS AURELIUS CORNELIUS CELSUS

(25 BC-AD 50) was a Roman writer on medicine and surgery. He wrote several works, of which only one remains entire, his treatise De Medicina in eight books. Probably lived in Gallia Narbonensis.

(ur. ok. 25 p.n.e., zm. ok. 50), pisarz rzymski w medycynie i chirurgii. Napisał wiele dzieł, z których tylko jedno pozostaje kompletne jego traktat De Medicina w ośmiu książkach. Mieszkał prawdopodobnie w Gallia Narbonensis.



Figure 1. Aulus Aurelius Cornelius Celsus

CAMP-FEVER SIGN – TYPHUS FEVER

Epidemic typhus (also called "camp fever", "jail fever", "hospital fever", "ship fever", "famine fever", "putrid fever", "petechial fever", "Epidemic louse-borne typhus" and "louse-borne typhus") is a form of typhus so named because the disease often causes epidemics following wars and natural disasters. The causative organism is Rickettsia prowazekii, transmitted by the human body louse (*Pediculus humanus corporis*).

OBJAW GORĄCZKI OBOZOWEJ - TYPHUS

Tyfus epidemiczny (zwany także "gorączka obozowa", "gorączka więzienna", "gorączka szpitalna", "gorączka okrętowa", "gorączka głodu", "tyfus", "wybroczyny gorączkowe", "epidemiczne wszy przenoszące tyfus", "wszy przenoszące tyfus") te odmiany są tak określone, ponieważ choroba często powoduje epidemie podczas wojen oraz katastrof naturalnych. Drobnostrój Rickettsia prowazekii są przekazywane przez wszy na ludzkie ciała (*Pediculus humanus corporis*).

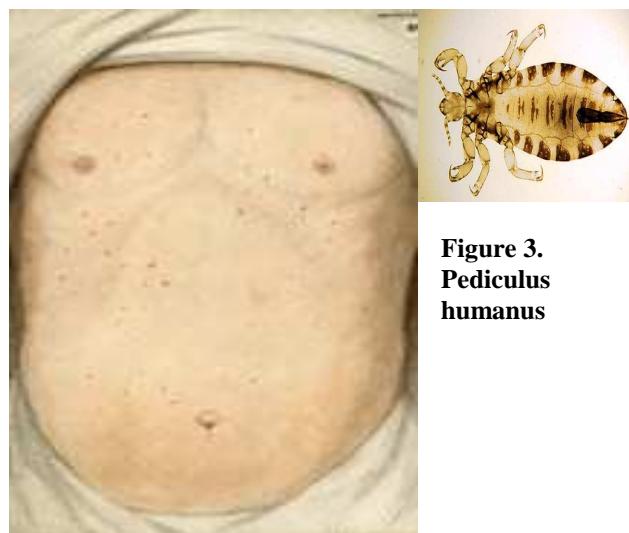


Figure 2. Camp-Fever sign

Figure 3.
*Pediculus
humanus*

CARO-SENEARA LESIONS

A pattern of psoriasis that should be distinguished from dermatitis. These depressed plaques are often on the sides of the fingers or border of the hand and have central umbilication.

LINIE CARO-SENEARA

Wzór łuszczycy, który powinien być dystingowany (wyróżniony) od stanu zapalnego skóry. Te depresje występują często na bocznych powierzchniach palców lub na granicy dłoni, z centralnym zagłębiem.

MARCUS R. CARO

1951-1960. Directors and consultants American Board of Dermatology. Instructor in Dermatology and Syphilology in the Medical School, 1915-1916, Chicago. Dr. Caro was recognized for his outstanding teaching

skills and had chaired many post-graduate courses and seminars in dermatopathology.



Figure 4. Marcus R. Caro

1951-1960. Dyrektor i konsultant American Board of Dermatology. Członek Dermatologii i Syfilologii w Medical School, 1915-1916, Chicago, USA. Dr Caro został uznany za wybitne umiejętności dydaktyczne i przewodził wieloma podyplomowymi kursami i seminariami z Dermatopatologii.

FRANCIS E. SENEAR

1889-1958. In his long academic career, Dr. Senear served in many leadership positions of a variety of dermatology societies including chairman of the Sections of Dermatology of the American Medical Association and the Illinois State Medical Society, president of the Chicago Dermatological Society (1927), president of the American Dermatological Association (1938), president of the American Board of Dermatology (1946-1949), and president of American Academy of Dermatology (1955). Dr. Senear has authored many dermatological articles and the most noticeable was his article entitled "An unusual case of pemphigus combining features of lupus erythematosus" which subsequently coined the term "Senear-Usher Syndrome" and this unique disease entity is now recognized as pemphigus erythematosus.



Figure 5. Francis E. Senear

1889-1958. W swojej długiej karierze naukowej dr. Senear występował w wielu stanowiskach kierowniczych w różnych towarzystwach dermatologicznych, w tym przewodniczył Sekcji Dermatologii American Medical Association oraz Illinois State Medical Society, był prezesem Towarzystwa Dermatologicznego Chicago (1927), prezesem Amerykańskiego Towarzystwa Dermatologicznego (1938), prezesem American Board of Dermatology (1946-1949) i prezesem American Academy of Dermatology (1955). Dr Senear jest autorem wielu artykułów dermatologicznych najbardziej doceniony został jego artykuł zatytułowany "nietypowy przypadek pęcherzycy mającej cechy tocznia rumieniowatego", który następnie wprowadził termin "Senear-Usher Syndrome" i ta wyjątkowa jednostka chorobowa jest obecnie uznawana za pęcherzyce rumieniowata.

CARPET-TACK SIGN

Discoid lupus erythematosus is a form of cutaneous lupus that presents as erythematous, hyperkeratotic, scaly plaques on sun-exposed areas. It is usually seen in young adults, with women affected twice as frequently as men. When the edges of these plaques are pulled back, the undersurface may look like that of carpet tacking.

OBJAW FASTRYGOWANEGO DYWANU

(fastrygować: zszywać, z reguły niedbale, przed właściwym szyciem)
Toczeń rumieniowaty ogniskowy jest formą skórną tocznia rumieniowatego, który przedstawia się jako hiperkeratotyczne, łuszczące się blaszki na obszarach eksponowanych na słońce. Zazwyczaj występuje u osób młodych, u kobiet dwa razy częściej niż u mężczyzn. Gdy krawędzie tych blaszek są odciągane, dolna powierzchnia może wyglądać podobnie, jak fastrygowany dywan.

CARRIÓN'S SIGN

synonym - verruca peruana, Peruvian wart, haemorrhagic pian. A soft conical or pedunculated papule that erupts in groups as a manifestation of the second stage of Bartonellosis. Anywhere on the skin or mucous membranes from miliary size to several centimeters.

OBJAW CARRIÓNIA

synonym: brodawka peruwiańska, verruca peruana, Peruvian wart, haemorrhagic pian. Miękkie lub stożkowate nieuszypułowane grudki, które wyrastają w grupach jako przejaw drugiego etapu Bartonellozy. Pojawiają się w dowolnym miejscu na skórze lub błonach śluzowych od prosówkowej wielkości do kilku centymetrów.

DANIEL ALCIDES CARRIÓN

Peruvian medical student, born 1850, Cerro de Pasco, died October 5, 1885.

As a new factor of the problem was a noticeable increase in verruca peruana. This disease, which manifests with wart-like skin eruptions of various shapes and sizes, had been present in Peru already in pre-Columbian times. From 1881 he conducted extensive research on verruga peruana, including clinical studies at the Dos de Mayo hospital in Lima. Carrion recognised that the disease was endemic, but not contagious, and that it was caused by an "agente verrucoso", possibly by a parasite attacking the blood and destroying leucocytes. In order to find out whether the disease could be inoculated and to study its clinical course, Carrón decided to conduct an experiment on himself. On August 27, 1885, Carrion took blood from a redly coloured verruca in the area of the eyebrows from a 14 year old boy. Carrion experienced the first symptoms of the disease on September 17, on October 5 he succumbed to the disease. The Universidad Nacional Daniel Alcides Carrión in Pasco, Peru, is named for him. Carrón cost him his life proving that, in fact, Oroya fever, and warts Peruvian are two manifestation of the same infectious diseases, today often referred to as a Carrón disease or bartonellosis. In 1909, Alberto Barton has detected the pathogen causing the disease, later called Bartonella bacilliformis.



Figure 6. Daniel Alcides Carrón

Peruwiański student medycyny, urodzony 1850, Cerro de Pasco, zmarł 05 października 1885.

Obserwowałauważalny wzrost verruca peruana. To choroba, która objawia się brodawkowatymi wykwitami skórnymi o różnych kształtach i rozmiarach, była obecna w Peru już w czasach prekolumbijskich. Od 1881 roku przeprowadził szeroko zakrojone badania nad verruca peruana, w tym badania kliniczne w szpitalu de Dos Mayo w Limie. Carrion uznał, że choroba miała charakter endemiczny, ale nie jest zaraźliwa, i że była spowodowana przez "agente verrucoso", ewentualnie przez pasożyta atakującego i niszczącego leukocyty we krwi. Aby dowiedzieć się, czy choroba może być

zaszczepiona i zbadać jej przebieg kliniczny, Carrión postanowił przeprowadzić eksperyment na sobie. 27 sierpnia 1885, Carrion pobrął krew z brodawki okolicy brwi od 14-letniego chłopca. Carrion doświadczył pierwszych objawów choroby w dniu 17 września, w dniu 5 października uległ chorobie i zmarł. Universidad Nacional Daniel Alcides Carrión w Pasco, Peru, jest nazwany jego imieniem. Carrión przypłacił życiem dowiedzenie, że w istocie gorączka Oroya i brodawka peruwiańska stanowią dwie manifestacje tej samej choroby zakaźnej, dziś często określanej jako choroba Carrióna albo Bartonellosza. W 1909 roku Alberto Barton wykrył patogen wywołujący chorobę, nazwany później *Bartonella bacilliformis*.

CARROT SIGN – CAROTENEMIA

The yellow pigmentation of the skin from excess carotene intake. Also associated with mangoes, pawpaw and oranges. May indicate a defect in the enzymatic conversion of vitamin A. Also seen in hyperbetaipoproteinaemia.



Figure 7. Carrot sign

OBJAW MARCHEWKI - KAROTENEMIA

Żółte zabarwienie skóry spowodowane nadmiernym spożyciem karotenu. Również związane ze spożyciem mango, papai i pomarańczy. Zmiany mogą wskazywać na wadę w enzymatycznej konwersji witaminy A. Także spotykane w hiperbetaipoproteinemii.

CARTER'S SIGN – Asiatic relapsing fever

Infection characterized by one or more attacks of fever beginning and ending abruptly and separated by an afebrile period of varying duration. The disease is prevalent in many parts of the world. The causative organism is a spirochete of the genus *Spirochaeta* (*Borrelia*) and transmission is by infected lice or ticks. Occurs in places where poverty and overcrowding predispose to human infestation with lice, during wars, when good hygiene is impossible.

OBJAW CARTERA - Azjatycka gorączka nawracająca

Zakażenie charakteryzuje się jedną lub kilkoma atakami gorączki rozpoczęjącej i kończącej się nagle, o różnym czasie trwania. Choroba jest powszechna w wielu

częściach świata. Drobnoistroje, krętki z rodzaju *Spirochaeta* (*Borrelia*) są transmitowane przez zakażone wszy i kleszcze. Choroba występuje w miejscach gdzie jest ubóstwo i przeludnienie, w czasie wojen, gdy prawidłowa higiena jest niemożliwa.

HENRY VANDYKE CARTER

Anglo-indian physician – (22. may 1831-4. may 1897). Anatomist, surgeon, and anatomical artist most notable for his illustrations of the book, Gray's Anatomy. He was formally educated at Hull Grammar School before moving to London to study medicine at St. George's Hospital. In 1852 he became a member of the Royal College of Surgeons in June 1853 where he began studying human anatomy and comparative anatomy. In January 1858 he joined the Bombay Medical Service as an assistant surgeon (assistant surgeon), and in March of this year came to India. He was assigned to the middle of the armed forces of India and sent to the town of Mhow. In May 1858 he became professor of anatomy and physiology of the Grant Medical College. In 1870 he was promoted to surgeon in the same year a surgeon major, lieutenant colonel in 1878, the (surgeon-lieutenant-colonel) in 1882, he was commander of the brigade (brigade-surgeon). He studied leprosy and leishmaniasis patients (then known under various names, such as an from Aleppo ulcer) in Italy, Greece, Algeria, Crete, Syria and Anatolia. Working in India dealt with tropical medicine and made her the many discoveries. During the great famine in India (1877-1888) he discovered the pathogen that causes a fever from the bite of rat (*Spirillum minus*). Previously presented a classic description of the fungal disease known as mycetoma (a term introduced to medicine).

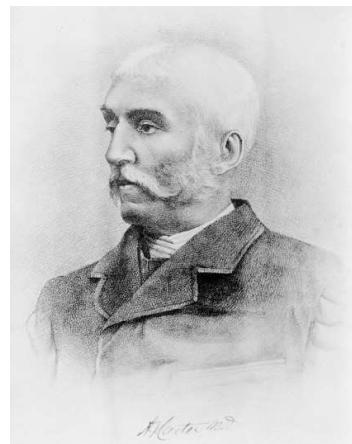


Figure 8. Henry Vandyke Carter

Anglo-hinduski lekarz (22.05.1831-4.05.1897). Anatom, chirurg i rysownik, twórca ilustracji do Gray's Anatomy. Pobierał nauki w Hull Grammar School, po czym przeniósł się do Londynu, gdzie studiował medycynę w St. George's Hospital. W 1852 został członkiem Royal College of Surgeons i w czerwcu 1853 roku rozpoczął tam studia anatomii człowieka i anatomii porównawczej. W styczniu 1858 roku wstąpił do Bombay Medical Service jako chirurg-asystent (assistant surgeon), w

marcu tego roku dotarł do Indii. Został przydzielony do sił zbrojnych śródkowych Indii i wysłany do miasta Mhow. W maju 1858 został profesorem anatomii i fizjologii Grant Medical College. W 1870 otrzymał awans na chirurga, w tym samym roku na chirurga majora, w 1878 na podpułkownika (surgeon-lieutenant-colonel), w 1882 został dowódcą brygady (brigade-surgeon). Badał trędowatych i chorych na leiszmaniozę (znaną wówczas pod różnymi nazwami, m.in. jako wrzód z Aleppo) we Włoszech, Grecji, Algierii, na Krecie, w Syrii i Anatolii. Pracując w Indiach zajmował się medycyną tropikalną i poczynił na jej polu wiele odkryć. Podczas wielkiej epidemii głodu w Indiach (1877-88 r.) odkrył patogen wywołujący gorączkę od ugryzienia szczury, śrubowca mniejszego (*Spirillum minus*). Wcześniej przedstawił klasyczny opis choroby grzybiczej, znanej jako stopa madurska albo mycetoma (który to termin wprowadził do medycyny).

CASAL'S NECKLACE SIGN

Lesions in the neck associated with hypersensitivity to sunlight in the course of Pellagra. Pellagra is a nutritional wasting illness caused by a deficiency of niacin (Vitamin B3) and tryptophan, in the body.



Figure 9. Casal's necklace sign



Figure 10. Casal's necklace sign

OBJAW NASZYJNIKA CASALA

Zmiany na szyi związane z nadwrażliwością na światło słoneczne w przebiegu Pellagry. Pellagra jest chorobą wyniszczającą spowodowaną niedoborem niacyny (vitamina B3) i tryptofanu, w organizmie.

DON GASPAR CASAL

Spanish physician (1691-1759). Casal was known as the "Spanish hippocrates" and was physician to King Ferdinand. His book on pellagra was published 3 years after his death. Pellagra was first identified among Spanish peasants by Don Gaspar Casal in 1735. A loathsome skin disease, it was called "mal de la Rosa" and often mistaken for leprosy.

Hiszpański lekarz (1691-1759). Casal był znany jako "Hiszpański Hipokrates" i był lekarzem Króla Ferdynanda. Jego książka o pellagrze została opublikowana 3 lata po jego śmierci. Pellagra po raz pierwszy została zidentyfikowana wśród hiszpańskich chłopów przez Don Gaspar Casala w 1735 roku. Odrażająca choroba skóry, była nazywana „mal de la Rosa” i często mylona z trądem.

CAT'S SIGN – murine typhus

Murine typhus (also called endemic typhus) is a form of typhus transmitted by fleas (*Xenopsylla cheopis*), usually on rats. (This is in contrast to epidemic typhus, which is usually transmitted by lice. Most people who are infected do not realize that they have been bitten by fleas. It is caused by the bacteria *Rickettsia typhi*, and is transmitted by the fleas that infest rats. Less often, endemic typhus is caused by *Rickettsia felis* and transmitted by fleas carried by cats or opossums. Symptoms may resemble those of measles, rubella, or possibly Rocky Mountain spotted fever. Murine typhus is found most commonly in southern California, Texas and Hawaii.

OBJAW KOTA

Tyfus myszy (tzw. tyfus endemiczny) jest formą tyfusu przenoszonego przez pchły (cheopis *Xenopsylla*), zwykle przez szczury. Jest to przeciwnieństwo epidemii tyfusu, który jest zwykle przekazywany przez wszy. Większość osób zakażonych nie zdaje sobie sprawy, że zostały one ugryzione przez pchły. Jest on spowodowany przez bakterie *Rickettsia typhi*, i jest przenoszony przez pchły, które opanowały szczury. Rzadziej endemiczny tyfus jest spowodowany przez *Rickettsia felis* i przekazywany przez pchły pochodzące od kota lub oposów. Objawy mogą przypominać odrę, rózyczkę, ewentualnie gorączkę plamistą Górz Skalistych. Tyfus myszy najczęściej występuje w południowej Kalifornii, Teksasie i na Hawajach.

CAT SCRATCH SIGN

Lymphadenopathy and sepsis from the zoonotic Bartonella bacteria found in cats. After first being identified in 1985, *Rochalimaea henselae*, later reclassified as *Bartonella henselae*, was determined conclusively to be the primary organism causative of catscratch disease. Dr. Robert Debré was the first to recognize the cat as a vector for this disorder and coined the term "catscratch" disease and sign in 1931.



Figure 11,12,13. Cat scratch sign

OBJAW ZADRAPANIA PRZEZ KOTA

Powiększenie węzłów chłonnych i posocznica wywołane odzwierzętą, chorobotwórczą bakterią *Bartonella*, znaną u kotów. Na początku identyfikowano w 1985 roku, *Henselae Rochalimaea*, później zmieniono nazwę na *Bartonella Henselae* i ustalono ostatecznie jako podstawowy czynnik sprawczy tego objawu / choroby. Dr Robert Debré pierwszy rozpoznał kota jako wektor dla tego zaburzenia i wprowadził termin choroby i objawu „catscratch” w 1931 roku.

ROBERT DEBRÉ

(1882, Sedan, Ardeny-1978) was a French physician (pediatrician). He gave his name to the most important pediatric hospital in Paris, France. A member of the Académie de Médecine. In 1946, he wrote with Pr. Paul Rohmer a famous manual entitled "Traité de Pathologie Infantile" (2500 pages, 2 volumes) which became a reference for a whole generation of pediatricians.



Figure 14. Robert Debré

(1882, Sedan, Ardeny-1978), francuski lekarz (pediatra). Imieniem jego nazwano najważniejszy szpital pediatryczny w Paryżu, Francja. Członek Académie de Médecine. W 1946 roku napisał z Pr. Paul Rohmerem słynny podręcznik zatytułowany "Traité de Pathologie Infantile" (2500 stron, 2 tomy), który stał się punktem odniesienia dla całego pokolenia pediatrów.

CAUTERY SIGNS

Often circular burn marks over areas of long standing pain. These areas have been burned as a form of primitive medical treatment for the condition. Burns on the hands and arms can mimic melanoma. There may be burns on the abdomen, back, and extremities. On the skull these cautery burns are sometimes in the form of a cross, inflicted as a treatment for headaches and fevers in childhood. In West Africa infants and children with febrile convulsions may be treated by plunging their feet into a cooking pot of boiling oil, causing horrific burns.

OBJAW PRZYŻEGANIA

Często okrągły blizny na obszarach o stałym, długo utrzymującym się bólu. Obszary te zostały spalone jako forma prymitywnego leczenia choroby. Obszary spalenia na rękach i ramionach mogą naśladować czerniaka. Mogą być też oparzenia brzucha, pleców i kończyn. Na czaszce te oparzenia wypalanie są czasami w formie krzyża, zadane w leczeniu bólu głowy i gorączki u dzieci. W Afryce Zachodniej drgawki gorączkowe niemowląt i dzieci mogą być leczone przez zanurzenie nogi w gotującym się na kuchni garnku z wrzącym olejem, powodując przerażające poparzenia.

CAYENNE PEPPER PUS SIGN

Cayenne-pepper granules within drops of pus. A sign indicating actinomycosis.



Figure 15. Cayenne pepper pus sign

OBJAW GRANULEK PIEPRZU CAYENNE

Granulki pieprzu Cayenne w kropli ropy. Objaw wskazujący na promienice.

CHADWICK'S SIGN

Symptom seen in early pregnancy (6-8 weeks) consisting of bluish coloration of vaginal mucosa and vaginal part of the cervix. Is dependent on increased, due to pregnancy, blood supply to these areas, which leads to venous stasis. It belongs to the so-called probable signs of pregnancy (symptoms suggestive of pregnancy, however, does not allow her diagnosis) and is found during pelvic examination.

OBJAW CHADWICKA

Objaw widoczny we wczesnej ciąży (6-8 tygodni) polegający na sino-purpurowym zabarwieniu błony śluzowej pochwy i części pochwowej szyjki macicy. Jest zależny od zwiększonego, spowodowanego ciążą ukrwienia tych okolic, który doprowadza do zastoju żylnego. Jest zaliczany do tak zwanych prawdopodobnych objawów ciąży (czyli objawów sugerujących ciążę, nie pozwalających jednak na jej rozpoznanie) i jest stwierdzany podczas badania ginekologicznego.

JAMES READ CHADWICK

(2 November 1844, Boston - 23 September 1905, Chocorua, New Hampshire) was an American gynecologist. Describing the Chadwick sign of early pregnancy in 1887. James Chadwick qualified with an M.D. from Harvard Medical School in 1871, and worked as a gynecologist in Boston. From 1871 to 1873 he studied obstetrics in Europe. From 1874 he worked at the Boston City Hospital. Became president of the American Gynaecological Society. He was a founder of the Boston Medical Library Association in 1875, and worked as the librarian until his death. He was voted president of the Association of Medical Librarians in 1904. He was the first president of the Harvard Medical Alumni

Association in 1891. He was a supporter of women in the practice of medicine. He died suddenly in 1905 at his summer home in New Hampshire, probably as a result of a fall from a piazza roof.



Figure 16. James Read Chadwick

(02.11.1844, Boston – 23.09.1905, Chocorua, New Hampshire), amerykański ginekolog. Opisał objaw Chadwicka, wczesnej ciąży w 1887 roku. James Chadwick ukończył Harvard Medical School w 1871 roku i pracował jako ginekolog w Bostonie. Od 1871 do 1873 studiował położnictwo w Europie. Od 1874 pracował w Boston City Hospital. został prezesem Amerykańskiego Towarzystwa Ginekologicznego. Był założycielem Boston Medical Library Association w 1875 roku i pracował jako bibliotekarz aż do śmierci. Został wybrany prezesem Stowarzyszenia Bibliotekarzy Medycznych w 1904 roku. Był pierwszym prezesem Harvard Medical Alumni Association w 1891 roku. Był zwolennikiem kobiet w praktyce medycznej. Zmarł nagle w 1905 r. w jego letnim domu w New Hampshire, prawdopodobnie w wyniku upadku z dachu.

CHAGAS-CRUZ SIGN

Erratic fever, hepatosplenomegaly, brain and heart involvement. Also known as South American zoonotic protozoal trypanosomiasis. Caused by exposure to fecal triatoma insects.



Figure 17. Chagas-Cruz sign

OBJAW CHAGAS-CRUZA

Gorączka narzutowa, powiększenie wątroby i śledziony, zajęcie mózgu i serca. Również znany w Ameryce Południowej jako chorobotwórcza trypanosomoza pierwotniakowa. Spowodowany narażeniem na kał z owadów Triatoma.

CARLOS JUSTINIANO RIBEIRO CHAGAS

Brazilian parasitologist and physician, 1879-1934. He discovered Chagas disease, also called American trypanosomiasis in 1909, while working at the Oswaldo Cruz Institute in Rio de Janeiro. After a brief stint as a medical practitioner in the hinterlands, Chagas accepted a position in the port authority of Santos, São Paulo, with the mission of fighting the malaria epidemic. There he introduced an innovation, which consisted in using pyrethrum, an insecticide, to disinfect households, with surprising success. His published work on this method served as the basis of prevention of malaria all over the world.

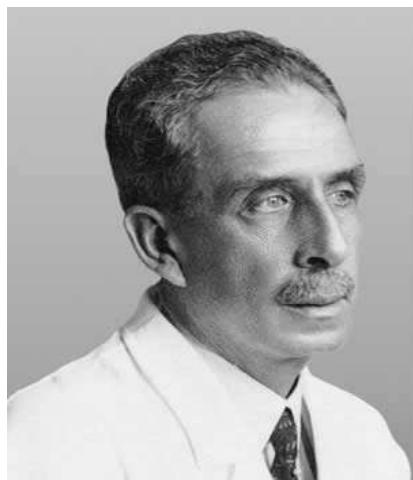


Figure 18. Carlos Justiniano Ribeiro Chagas

Brazylijski parazytolog i lekarz, 1879-1934. W 1909 roku odkrył, chorobę Chagasa, zwaną również trypanosomzą Amerykańską, podczas pracy w Oswaldo Cruz Institute w Rio de Janeiro. Po krótkim okresie praktyki lekarskiej w odległych rejonach, Chagas przyjął stanowisko w porcie Santos, São Paulo, z misją zwalczania epidemii malarii. Wprowadzono innowację, która polegała na użyciu złocienia, środka owadobójczego, do dezynfekcji gospodarstw domowych. Jego opublikowane prace na temat tej metody posłużyły jako podstawa profilaktyki malarii na całym świecie.

OSWALDO GONÇALVES CRUZ

Brazilian parasitologist and physician, 1871-1917. At the age of 15 he started to study at the Faculty of Medicine of Rio de Janeiro and in 1892 he graduated as medical. Inspired by the great work of Louis Pasteur, four years later he went to Paris to specialize in Bacteriology at the Pasteur Institute. Cruz was initially successful in the sanitary campaign against the bubonic plague, to which end he used obligatory notification of cases, isolation of sick people, treatment with the sera produced at Manguinhos and extermination of the rats populating the

city. On June 9, 1904, following a proposal by Oswaldo Cruz, the government presented a bill to the Congress requesting the reestablishment of obligatory smallpox vaccination. In 1907, on occasion of the 14th International Congress on Hygiene and Demography in Berlin, he was awarded with the gold medal in recognition of the sanitation of Rio de Janeiro.



Figure 19. Oswaldo Gonçalves Cruz

1879-1934. Brazylijski lekarz. W wieku 15 lat rozpoczął studia na Wydziale Lekarskim w Rio de Janeiro, w 1892 roku uzyskał dyplom lekarza medycyny. Zainspirowany przez wielkie dzieło Ludwika Pasteura, cztery lata później wyjechał do Paryża na specjalizację w dziedzinie bakteriologii w Instytucie Pasteura. Cruz odniósł sukces w kampanii przeciwko dżumie, w której to celu użył obowiązkowej notyfikacji przypadków, izolacji chorych, leczenia serum produkowanego w Manguinhos i eksterminacji szczurów w zaludnionym mieście. W dniu 9 czerwca 1904 r., na wniosek Oswaldo Cruz, rząd przedstawił projekt ustawy Kongresu z o przywrócenie obowiązkowego szczepienia przeciwko ospie. W 1907 roku z okazji 14. Międzynarodowego Kongresu Higieny i Demografii w Berlinie, został odznaczony złotym medalem w uznaniu za sanitarnie dokonania z Rio de Janeiro.

CHAGRES' SIGN

Synonym: Chagres River, Panama; L. febris. Malarial fever in Panamanian railroad workers. Chagres, a village of the Republic of Panama in the Colón Province.

OBJAW CHAGRES

Synonim: Chagres River, Panama; L. febris. Malaria u panamskich kolejarzy. Chagres to wieś w Republice Panamy leżąca w prowincji Colón.

CHARCOT'S SIGN

1. A sign of peripheral facial paralysis. 2. Intermittent limping. A sign of arteriosclerosis of the feet and legs. 3. Rareyng osteitis of a joint associated with tabes dorsalis.



Figure 20,21. Charcot's sign



Figure 22. Charcot's sign

OBJAW CHARCOTA

1. Objaw obwodowego porażenia mięśni twarzy. 2. Przerywane kuśtykanie. Objaw arteriosklerozy kończyn dolnych. 3. Rozrzedzające zapalenie kości i stawów związane z tabes dorsalis.

JEAN MARIE CHARCOT

1825-1893. French neurologist and professor of anatomical pathology. He is known as "the founder of modern neurology" and is "associated with at least 15 medical eponyms", including Charcot-Marie-Tooth disease and amyotrophic lateral sclerosis (Lou Gehrig's disease). His work greatly influenced the developing fields of neurology and psychology. He was the "foremost neurologist of late nineteenth-century France" and has been called "the Napoleon of the neuroses". He named and was the first to describe multiple sclerosis. He was also the first to describe a disorder known as Charcot joint.



Figure 23. Jean Marie Charcot

1825-1893. Francuski neurolog, profesor anatomii patologicznej. Jest znany jako "ojciec współczesnej neurologii" i jest "związany z co najmniej 15 eponimami medycznymi", w tym chorobą Charcot-Marie-Tooth i stwardnieniem zanikowym bocznym (choroba Lou Gehriga). Jego twórczość miała ogromny wpływ na rozwój neurologii i psychologii. Był "głównym neurologiem późnej XIX-wiecznej Francji" i nazywany był "Napoleonem nerwic". Nazwał i jako pierwszy opisał stwardnienie rozsiane. Był też pierwszym, który opisał zaburzenie znane jako stawy Charcota.

CHICKEN CHOLERA SIGN

A zoonotic disease caused by the *Pasteurella* bacterium. A contagious disease of fowls. Can cause septicaemic plague in humans. First described by Louis Pasteur. Opisany po raz pierwszy przez Ludwika Pasteura.



Figure 24. Chicken cholera sign

OBJAW CHOLERY KURCZAKÓW

Choroba odzwierzęca, spowodowana przez bakterie *Pasteurella*. Zaraźliwa choroba drobiu. Może powodować posocznicową plagę u ludzi.

LUDWIK PASTEUR

(December 27, 1822 – September 28, 1895) was a French chemist and microbiologist born in Dole. He is remembered for his remarkable breakthroughs in the causes and preventions of diseases. His discoveries reduced mortality from puerperal fever, and he created the first vaccine for rabies and anthrax. Work on diseases included work on chicken cholera. Both Institute Pasteur and Université Louis Pasteur were named after him.



Figure 25. Ludwik Pasteur

(27 grudnia 1822 - 28 września 1895), francuski chemik i mikrobiolog urodzony w Dole. Jego badania to przełom w przyczynie i prewencji chorób. Jego odkrycia zmniejszyły umieralność poporodowej gorączki i odkrył pierwszą szczepionkę przeciwko wściekliźnie i wąglikowi. Prowadził prace na temat cholery kurczaków. Zarówno Instytut Pasteura i Université Louis Pasteur, nazwano jego nazwiskiem.

CHLORACNE SIGN

Chloracne of the face as an indication of dioxin poisoning.

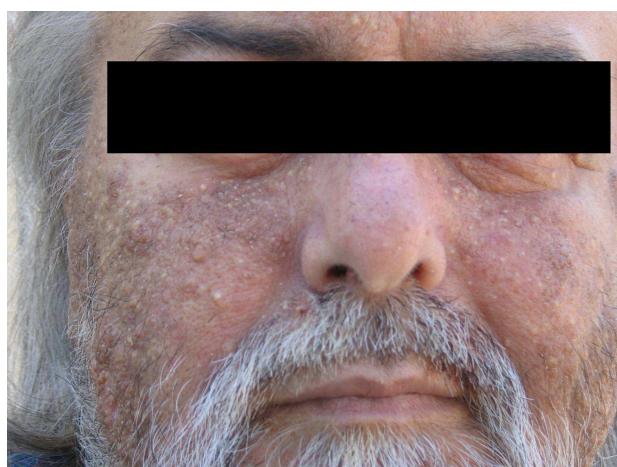


Figure 26. Chloracne sign

OBJAW CHINSKIEGO DZIOBA PO OSPIE

Tradzik chlorowcowy na twarzy, jako wskaźnik zatrucia dioksynami.



Figure 27,28. Chloracne sign

CHINA POCKMARK SIGN

(before c. 1700, China). Eastern folkways ritual smallpox inoculations made between the thumb and forefinger resulting with pockmark scar.

OBJAW CHINSKIEGO DZIOBA PO OSPIE

(przed 1700 r., Chiny). Wschodnie rytmalne zwyczaje szczepienia ospy pomiędzy kciukiem i palcem wskazującym, ukazujące się jako blizna w kształcie dzioba.

CHLOROSIS SIGN

While mottling of the hair extending two inches from the head in a patient sick with chlorosis. Chlorosis, an affliction of young women through the ages, has recently disappeared from the records of Medicine. Victims of chlorosis were usually maidens in their middle teens. Physically they always seemed well-nourished. Their skin, however, had a greenish-yellow tinge, especially in brunettes. Such chlorotic girls constantly complained of being tired. Egyptians 3,500 years ago suffered from an "AAA disease" which resembled chlorosis. In the Middle Ages doctors called it morbus virgineus (virgin's disease). Shakespeare called it greensickness. Probably

the most logical view was a long-continued iron deficiency in the diet. (Richelot c. 1800).

OBJAW CHLOROZY

Plamistość włosów (nakrapiane włosy) w odległości dwacale od głowy u pacjenta z chlorozą. Chloroza, schorzenie młodych kobiet przed wiekami, niedawno zniknęła z ewidencji chorób. Ofiarami chlorozy były zwykle dziewczęta w wieku kilkunastu lat. Fizycznie zawsze wydawały się dobrze odżywione. Ich skóra, jednak miała zielonkawo-żółty odcień, zwłaszcza u brunetek. Takie chlorotyczne dziewczyny ciągle skarzyły się na zmęczenie. Egipcjanie 3500 lat temu cierpieli na chorobę "AAA", która przypominała chlorozę. W średniowieczu lekarze opisywali „morbus virgineus” (pierwotna choroba). Szekspir nazwał ją „blednicą”. Prawdopodobnie najbardziej logiczną przyczyną był długotrwały niedobór żelaza w diecie. (Richelot ok. 1800 rok).

LOUIS GUSTAVE RICHELOT

(1806 – September 1893) was a French physician born in Nantes. In 1831 he earned his doctorate in Paris with the dissertation, *De la uterine phlébite*. During his career he worked as a dispensary physician and for the Bureaux de bienfaisance. He is remembered for providing French translations of English medical works



Figure 29. Louis Gustave Richelot

(1806 - wrzesień 1893), francuski lekarz, urodził się w Nantes. W 1831 uzyskał doktorat w Paryżu z pracy doktorskiej, *De la uterine phlébite*. W trakcie swojej kariery pracował jako lekarz w poradni Bureaux de bienfaisance. Zapisał się w pamięci jako tłumacz francuski prac medycznych na język angielski.

CHOJNOWSKI'S SIGN

Chromidrosis, perspiralion with the color of and consistency of milk (Chojnowski 1863).

OBJAW CHOJNOWSKIEGO

Chromidrosis, podobne do koloru i konsystencji mleka (Chojnowski 1863).

BRONISŁAW CHOYNOWSKI (CHOJNOWSKI)

(3 May 1836 Murzyńce (Ukraine) - April 6, 1870). Chojnowski graduated with a gold medal, and the medical department at the Kiev University with honors (1858). Medical career began in Kiev, first as an assistant at the clinic as therapeutic, and later an active member of the Society of Doctors of Kiev. Experience gained in the clinic was the subject of several articles published in professional journals Kiev (eg "Sowremiennoja medicina"). Received his doctorate in 1863 for his dissertation *The diurnal temperature variation in healthy and diseased human*. It was based not only on clinical experience, conducted on patients, since the first few months Chojnowski examined his body temperature fluctuation, "spent sleepless nights with a thermometer in his hand". In the same year 1863, he left for further studies in Krakow, Prague, Berlin, Vienna, and even Paris, where he practiced in the local hospitals. In Warsaw, at the Central School in 1865 was awarded the title of assistant professor and assistant professor of pathology and therapy in detail. At the same time began at the university lectures in dermatology. In 1867 he received the title of professor lecturer. Very active in the medical community: he was one of the founders of "Gazeta Physicians" (1866) and a member of the Medical Society of Warsaw, Krakow and Prague. Bronisław Chojnowski, above all deal with dermatology and research on fluctuations in body temperature (as the first began to use medical thermometer.) He authored 14 research papers in these areas and feature a short history of dermatology. Doctor on duty at the university clinic, contracted typhus and died at the age of 34 years.



Bronisław Chojnowski. (Poług fotografii Witkowskiego).

Figure 30. Bronisław Chojnowski

(3 maja 1836 r., Murzyńce (Ukraina) - 6 kwietnia 1870 r.). Chojnowski ukończył wydział lekarski na Uniwersytecie Kijowskim z wyróżnieniem (1858 r.). Karierę lekarską rozpoczętał w Kijowie, najpierw jako asystent w klinice terapeutycznej, a z czasem aktywny działacz Towarzystwa Lekarzy Kijowskich. Doświadczenie zdobywane w klinice stało się przedmiotem kilku artykułów, publikowanych w kijowskiej prasie fachowej (m.in. "Sowremiennoja medicina"). Tytuł doktora medycyny uzyskał w 1863 r. za rozprawę „O dobowym wahaniu temperatury u

człowieka zdrowego i chorego". Opierała się ona nie tylko na doświadczeniach klinicznych, przeprowadzonych na pacjentach, gdyż w pierwszej kolejności Choynowski przez kilka miesięcy badał wahania temperatury swojego ciała "bezsennie spędzając nocę z termometrem w ręku". W tymże roku 1863 r. wyjechał na dalsze studia do Krakowa, Pragi, Berlina, Wiednia, a nawet Paryża, gdzie praktykował w tamtejszych szpitalach. W Warszawie, w Szkole Głównej w 1865 r. uzyskał habilitację oraz tytuł docenta patologii i terapii szczegółowej. Jednocześnie rozpoczął na tej uczelni wykłady z dermatologii. W 1867 r. otrzymał tytuł profesora adiunkta. Bardzo aktywnie działał w środowisku lekarskim: był jednym z założycieli "Gazety Lekarskiej" (1866 r.) oraz członkiem Towarzystwa Lekarskiego Warszawskiego, Krakowskiego i Praskiego. Bronisław Chojnowski zajmował się przede wszystkim dermatologią i badaniami nad wahaniem temperatury ciała (jako pierwszy zaczął stosować termometr lekarski). Był autorem 14 prac naukowych z wspomnianych dziedzin oraz krótkiego rysu historii dermatologii. Pełniąc służbę lekarską w klinice uniwersyteckiej, zaraził się tyfusem i zmarł w wieku 34 lat.

CHRISTMAS TREE

Distribution of the rash of pityriasis rosea.



Figure 31. Christmas tree

OBJAW CHOINKI

Dystrybucje zmian skórnych w przebiegu łupieżu różowego.

CIRRHOSIS BACK SIGN

Excoriations of the back secondary to scratching. An early sign of primary biliary cirrhosis.

OBJAW CIRRHOSIS BACK

Otarcia naskórka wtórnie do zadrapań. Wczesny objaw pierwotnej marskości żółciowej.

CLARKE'S TONGUE SIGN

A fissured indurated tongue due a syphilis.

OBJAW JĘZYKA CLARKE

Sztwardniała szczelina spodu języka w przebiegu kiły.

Sir CHARLES MANSFIELD CLARKE (1st Baronet)

English physician, 1782-1857. After leaving St Paul's School, he received his medical training at St George's Hospital and the Hunterian School of Medicine. He spent two years as assistant surgeon in the Hertfordshire Militia. He left the army and, specialised in midwifery and in women's and children's diseases. Between the years 1804 and 1821, he delivered regular courses of lectures on these subjects. His reputation as a practitioner during these years reached great heights and numerous honours were bestowed on him, including the Fellowship of the Royal Society in 1825, the appointment of Physician to Queen Adelaide in 1830, a baronetcy in 1831, and honorary degrees at Cambridge and Oxford in 1842 and 1845. He was president, and an enthusiastic supporter, of the Society for the Relief of the Widows and Orphans of Medical Men.



Figure 32. Charles Mansfield Clarke

Angielski lekarz, 1782-1857. Po wyjeździe z St Paul's School, otrzymał wykształcenie medyczne w St George's Hospital i Hunterian School of Medicine. Spędził dwa lata jako asystent chirurga w Hertfordshire Militia. Odszedł z armii i specjalizował się w zakresie położnictwa, chorób kobiecych i dziecięcych. W latach 1804 i 1821, wydawał regularne tomły wykładów na te tematy. Jego reputacja jako lekarza w ciągu tych lat osiągnęła wyżyny i nadano mu liczne wyróżnienia, w tym Fellowship of the Royal Society w 1825 roku, stanowisko lekarza królowej Adelajdy w 1830 roku, w 1831 roku Baroneta i doktora Honoris Causa w Cambridge Oxford w 1842 i 1845. Był prezesem i gorącym zwolennikiem, Towarzystwa Pomocy Wdów i Sierot po Medykach.

CLAVICULAR SIGN

A tumefaction at the inner third of the right clavicle; seen in congenital syphilis. It's an end result of neonatal periostitis. Also known as Higoumenaki's sign. Sign has

been described by Georgios Higoumenakis in 1927 on the pages of the Greek journal Πρακτικά Ιατρικής Εταιρείας Αθηνών (Reports of the Medical Society of Athens).



Figure 33. Clavicular sign

OBJAW OBOJCZYKOWY

Jednostronne guzowate zgrubienie po wewnętrznej, 1/3 stronie prawego obojczyka, spotykany w przebiegu kily wrodzonej. Jest to końcowy wyniki zapalenia okostnej u noworodków. Objaw znany również jako objaw Higoumenaki's. Objaw został opisany przez Georgiosa Higoumenakisa w 1927 roku na łamach greckiego czasopisma Πρακτικά Ιατρικής Εταιρείας Αθηνών (Sprawozdania Towarzystwa Medycznego w Atenach).

GEORGE HIGOUMENAKIS

(1895–1983) was a Greek dermatologist born in Iraklion of Crete (Greece). He studied medicine at the Medical School of the National University of Athens. He then chose to become a dermatologist and went to France to fulfil his desire. He was a student of Gaston Milian, a famous syphilologist, at the Hospital St. Louis. He returned to Greece in 1924, became a member of the Medical Society of Athens and began practicing medicine privately. He became a director of the Department of Dermatology at the hospital "Evaggelismos" and practiced medicine successfully until the 1940s.

(1895-1983) był greckim dermatologiem, urodził się w Iraklionie na Krecie (Grecja). Studiował medycynę w Szkole Medycznej Narodowego Uniwersytetu Ateńskiego. Następnie jako dermatolog udał się do Francji, by spełniać swoje pragnienia. Był uczniem Gastona Milian, sławnego syphilologa, w szpitalu St. Louis. Po powrocie do Grecji w 1924 roku, został członkiem Towarzystwa Lekarskiego w Atenach i rozpoczął prywatną praktykę lekarską. Był kierownikiem Katedry i Kliniki Dermatologii w szpitalu "Evaggelismos" i praktykował z powodzeniem aż do 1940 roku.



Figure 34. George Higoumenakis

CLUBBED FINGERS SIGN

Clubbing of the fingers and fingernails as a sign of chronic anorexia, cirrhosis of the liver, and bacterial endocarditis. The ends of the fingers may have the appearance of drum sticks. Also called Hippocratic fingers.



Figure 35,36. Clubbed fingers sign

OBJAW PALCÓW MACZUGOWATYCH

Maczugowate palce i paznokci jako przejaw przewlekłej anoreksji, marskości wątroby i bakteryjnego zapalenia wsierdzia. Końce palców mogą mieć wygląd drum sticks (pałeczki do gry na perkusji). Zwany również palcami Hipokratesa.

COBB'S SIGN

Sudden fever with pigmentation of the nose and cheeks seen in India.

OBJAW COBB-a

Nagła gorączka z pigmentacją nosa i policzków, spotykana w Indiach.

STANLEY COBB

(December 10, 1887 – February 25, 1968) was a neurologist and could be considered "the founder of biological psychiatry in the United States. In 1925 he was named Harvard's Bullard Professor of Neuropathology. In 1956, Cobb received the George M. Kober Medal for his contributions to medicine.



Figure 37. Stanley Cobb

(10 grudnia 1887 - 25 lutego 1968) neurolog i uważany również za "założyciela "Biological Psychiatry" w Stanach Zjednoczonych. W 1925 roku został mianowany profesorem Uniwersytetu Harwarda Bullard neuropatologii. W 1956 roku Cobb otrzymał „George M. Kober Medal” za zasługi dla medycyny.

COCHIN SIGN

Elephantiasis of the leg. Sign caused by *Wuchereria malayi*. Clarke in 1709 roku called elephantiasis of the legs in Cochin, South India "Malabar legs", (see Menon).



Figure 38. Cochin sign

OBJAW COCHINA

Słoniowacizna nóg. Objaw spowodowany przez *Wuchereria malayi*. Clarke w 1709 roku opisał słoniowaciznę nóg w mieście Cochin (tłum. Koczyn), Indie Południowe "nogi Malabara".

"CLUSTER OF JEWELS" SIGN

Cutaneous lesions of linear IgA bullous disease (LABD) are usually nonscarring blisters, often extensive on trunk and extremities. They are characterized by the "cluster of jewels" sign, with vesicles and bullae at edges of polycyclic lesions..



Figure 39,40. "Cluster of jewels" sign

OBJAW "KLASTEROWEJ KOLII"

Zmiany skórne w linijnej IgA chorobie pęcherzowej (LABD) przebiegają zazwyczaj z pęcherzami, bez pozostawiania blizn. Zmiany często są obszerne i występują na tułowiu i kończynach. Charakteryzują się one "zebranymi w grono" objawem, z pęcherzami różnej wielkości na krawędziach policyklicznych zmian.

COMBY SIGN

White patches of degenerated epithelium on the buccal mucous membrane and gingival tissues. An early sign of measles (rubeolla).



Figure 41. Comby sign

OBJAW COMBY

Białe plamy na zdegenerowanym nabłonku błony śluzowej jamy ustnej i dziąsłach. Wczesny objaw odry.

JULES COMBY

French paediatrician, 1858-1942. He published the influential *Traité des maladies de l'enfance* (Treatise of the Diseases of Childhood).



Figure 42. Jules Comby

Francuski pediatra, 1858-1942. Opublikował wpływowy *Traité des maladies de l'enfance* (Traktat z chorób dzieciństwa).

COMPLETE PTOSIS SIGN

Third cranial nerve palsy. A sign seen in cerebrospinal syphilis.

OBJAW CAŁKOWITEGO OPADANIA POWIEK

Porażenie III nerwu czaszkowego. Objaw spotykany w kile mózgowo-rdzeniowej.

COOL SIDE SIGN

Unilateral anhidrosis found with lung carcinoma. Caused by destruction of the unilateral superior cervical ganglion resulting with the inability to sweat on the affected side.

OBJAW CHŁODNEGO BOKU

Jednostronne anhidrosis w przebiegu raka płuca. Spowodowane przez jednostronne zniszczenie przedniego zwoju szyjnego i wynikające z niemożności wydzielania w tym miejscu potu.

COYOTE SIGN

A rare zoonotic Brucella, disease from coyotes and dogs.

OBJAW KOJOTA

Rzadka choroba odzwierzęca – Brucella, pochodząca od kojotów i psów.

CRAB TUBERCULOSIS SIGN

A zoonotic pulmonary disease that resembles tuberculosis and can sometimes have CNS and dermalologic involvement. Caused by the ingestion of undercooked crabs and crayfish containing the *Paragonimus* fluke.

OBJAW GRUŽLICY KRABOWEJ

Odzwierzęca płucna choroba przypominająca gruźlicę, a czasami może mieć objawy z Centralnego Układu Nerwowego i skóry. Spowodowany przez spożycie niedogotowanych krabów i raków zawierających przywry z rodzaju *Paragonimus*.

CORLETT'S SIGN

A contagiosa bullous form of impetigo beginning on the face..



Figure 43. Corlett's sign

OBJAW CORLETTA

Liszajec zakaźny rozpoczęjący się na twarzy.

WILLIAM THOMAS CORLETT

American dermatologist, 1854 – 1948. Introduced new methods to treat skin and venereal diseases, and researched the effect of climate, particularly cold, on skin diseases. He attended Oberlin College from 1870-73, and graduated with an M.D. from Wooster University Medical College of Wooster in 1877. In 1882 Corlett was appointed lecturer, then in 1884 professor, on skin and genito-urinary diseases at Wooster. In 1890, his title at WRU was changed to professor of dermatology and syphilology. As a member of the Board of Health in 1893, Corlett fought for better lighting and ventilation in public schools.

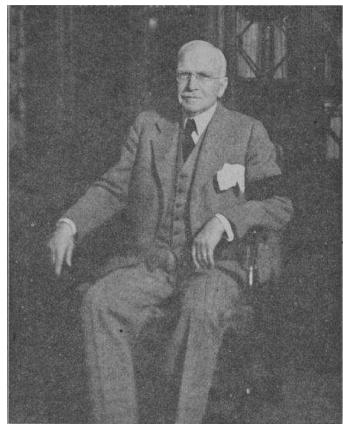


Figure 44. William Thomas Corlett

Amerykański dermatolog, 1854 – 1948. Wprowadził nowe metody w leczeniu skóry i chorób wenerycznych, badał wpływ klimatu, zwłaszcza zimna, na choroby skóry. Uczęszczał do Oberlin College w 1870/73, uzyskał dyplom z medycyny na University Medical College of Wooster w 1877 roku. W 1882 roku został mianowany wykładowcą na Uniwersytecie, a następnie w 1884 roku profesorem chorób skóry i układu moczowo-płciowego. W 1890 r. jego tytuł został zmieniony na profesora dermatologii i syfilologii. Jako członek Rady Zdrowia w 1893 roku, walczył o lepszą wentylację i oświetlenie w szkołach publicznych.

CORRIGAN SIGN

1. A purplish line between the gums and teeth, due to chronic copper intoxication. Syn. Corrigan Line. 2. A jerky carotid pulse characterized by full expansion followed by quick collapse

OBJAW CORRIGANA

1. Purpurowe linie między zębami a dziąsłami, powstałe w związku z przewlekłym zatruciem miedzią. Syn. Linie Corrigana. 2. Nierówne tępno na tętnicach szyjnych charakteryzujące się pełną ekspansją, a następnie szybkim załamaniem.

SIR DOMINIC JOHN CORRIGAN

Irish physician, born December 1, 1802, Dublin; died February 1, 1880, Dublin. Received his first medical

education in Dublin and then went to Edinburgh, where he received his doctorate in 1825. Corrigan returned to Dublin to open his own practice and subsequently became lecturer of medicine at the school of Diggs Street, Peter Street and Richmond Hospital. He was appointed physician to the Cork Street Fever Hospital, where he commenced his clinical-pathological work. In 1830 he became attached to "the Charitable Infirmary", Jervis Street Hospital, in Dublin. Despite the fact that he disposed of only six beds, he there conducted a series of pioneering experiments which have become famous on the symptomatology of heart disease. In 1856 he was elected member of the Irish College of Physicians (or: King and Queen's College of Physicians in Ireland?). He was also president of the Pathological Society of which he had been co-founder in 1838, and in 1875 became the first president of the Pharmaceutical society. Corrigan was created a baronet in 1866. Corrigan was responsible for the improvement of Dublin's water supply.



Figure 45. Dominic John Corrigan

Irlandzki lekarz, ur. 1 grudnia, 1802, Dublin, zmarł 01 lutego 1880 w Dublinie. Gdy ukończył swoją pierwszą edukację medyczną w Dublinie wyjechał do Edynburga, gdzie uzyskał doktorat w 1825 roku. Corrigan wrócił do Dublina, aby otworzyć własną praktykę i następnie został wykładowcą w szkole Diggs Street, Richmond i Peter Street Hospital. Został mianowany lekarzem w Cork Street Fever Hospital, gdzie rozpoczęła się jego kliniczno-patologiczna praca. W 1830 roku został dołączony do "Charitable Infirmary" Jervis Street Hospital w Dublinie. Pomimo tego, że posiadał tylko sześć łóżek, przeprowadzał tam serię pionierskich badań w kierunku chorób serca. W 1856 roku został wybrany członkiem irlandzkiego College of Physicians (lub: King i Queen's College of Physicians w Irlandii). Był także prezesem Towarzystwa Patologii której był współzałożycielem w 1838 roku, w 1875 został pierwszym prezesem Towarzystwa Farmaceutycznego. Corrigan otrzymał tytuł baroneta w 1866 roku. Corrigan był odpowiedzialny za poprawę zaopatrzenia w wodę w Dublinie.

CRAW CRAW SIGN

A form of pustular eczema found in West Africa.

OBJAW CRAW CRAW

Postać krostopowego wyprysku stwierdzony w Afryce Zachodniej.

CRESCENTIC NOTCH SIGN

There are depressions or notching of the incisal edges of the labial surfaces of the permanent incisors. A sign of congenital syphilis. Also called Hutchinson's Incisor sign and Screwdriver Teeth sign.

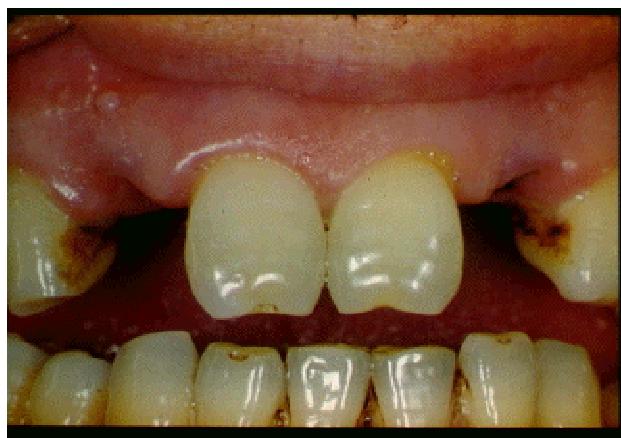


Figure 46. Crescentic notch sign

OBJAW PÓŁKOLISTYCH ZĘBÓW

Wgłębienia lub nacięcia krawędzi brzegu siekaczy na powierzchniach wargowych zębów stałych. Objaw kiły wrodzonej. Zwany również objawem siekaczy Hutchinsona lub objawem uzębienia w kształcie śrubokrętu.

SIR JONATHAN HUTCHINSON

English surgeon. 1828-1913. He received his professional qualification from Bartholomew's Hospital in 1850. During his student days in London Hutchinson became involved with philanthropic Quaker Missions, with the aim of alleviating misery and uplifting the impoverished. In 1851 he studied ophthalmology at Moorfields and was an ophthalmologist to the London Ophthalmic Hospital. He was also venereologist to the Lock Hospital, physician to the City of London Chest Hospital, and general surgeon to the London and Metropolitan Hospitals. Hutchinson developed a special interest in congenital syphilis, which was common in London in his time. It is said that he saw more than one million patients with syphilis in his lifetime. Hutchinson was a member of the Dermatological Society of London. He was a fellow of the Royal College of Surgeons from 1862 and professor of surgery there from 1879 to 1883. Hutchinson had a vast clinical experience and he published his observations in more than 1,200 medical articles. Produced the quarterly Archives of Surgery. In England the term *morbus Hutchinson-Boeck* has been

used for benign lymphogranulomatosis, now commonly known as Boeck's sarcoid. President of the Pathological Society 1879-1880, president of the Ophthalmological Society of Great Britain 1884-1885, president of the Royal College of Surgeons 1889, president of the Neurological Society 1887, president of the Medical Society of London 1892, president of the International Dermatological Congress 1896. For a brief period of time he was the editor of the British Medical Journal.

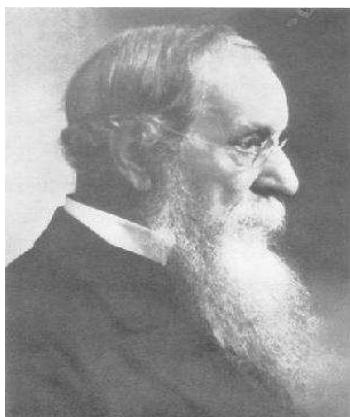


Figure 47. Jonathan Hutchinson

Angielski chirurg. 1828-1913. Uzyskał kwalifikacje zawodowe z Bartholomew's Hospital w 1850 roku. Podczas swoich studiów w Londynie Hutchinson związał się z misją dobrotową Quaker, w celu złagodzenia nędzy i pomóc ubogim. W 1851 studiował w Moorfields okulistykę i był okulistą w Londyńskim szpitalu Okulistycznym. Był również wenerologiem w Lock Hospital, lekarzem City of London Chest Hospital, chirurgiem ogólnym w Londynie w Metropolitan Hospitals. Hutchinson wykazywał specjalne zainteresowanie kiłą wrodzoną, która była powszechna w Londynie w tych czasach. Mówi się, że widział ponad milion pacjentów z kiłą w swoim życiu. Hutchinson był członkiem Londyńskiego Towarzystwa Dermatologicznego. Był członkiem Royal College of Surgeons od 1862 roku i profesorem chirurgii 1879/83. Hutchinson miał duże doświadczenie kliniczne i opublikował swoje spostrzeżenia w ponad 1200 artykułach medycznych. Redagował kwartalnik Archives of Surgery. W Anglii termin *morbus Hutchinson-Boeck* zostały wykorzystane do łagodnej lymphogranulomatozy, obecnie powszechnie znanej jako sarkoidozy Boeck's. Prezes Towarzystwa Patologii 1879-1880, Prezes Towarzystwa Okulistycznego Wielkiej Brytanii 1884-1885, przewodniczący Royal College of Surgeons 1889 roku, prezes Towarzystwa Neurologicznego w 1887 roku, Prezydent Medical Society of London 1892, Prezes Międzynarodowego Kongresu Dermatologii 1896 roku. Przez krótki okres czasu był redaktorem "British Medical Journal".

CROWE'S SIGN (AXILLARY FRECKLING)

Appears as multiple 1- to 4-mm freckling spots in the axillary vault and is seen in 25 to 50% of patients with neurofibromatosis.



Figure 48. Crowe's sign

OBJAW CROWE

Pojawienie się wielu 1 do 4 mm piegowatości w okolicy pach jest obserwowane u 25 do 50% pacjentów z nerwiakówkniakowatością.

CROWE FRANK W.

American physician (1919-1987).

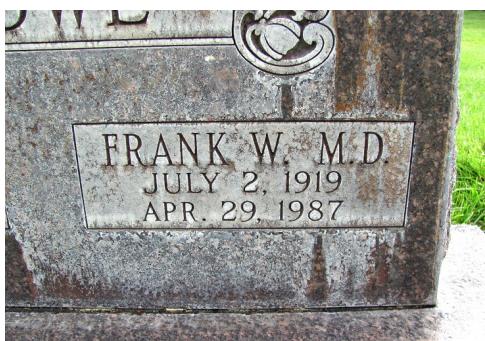


Figure 49. Crowe Frank W.

Amerykański lekarz (1919-1987).

CROWN OF VENUS SIGN

Papular lesions of secondary syphilis on the forehead near the hair margin.

OBJAW KORONY VENUS

Grudkowe zmiany w przebiegu wtórnej kiły na czole, w pobliżu linii włosów.

MIKHAIL AFANASIEVICH BULGAKOV

(1891–1940) was a Russian physician-writer whose doctor stories are based on his experience as a rural physician in a small village called Nikolskoye in the province of Smolensk.^{1(p8)} Nikolskoye was his first assignment after studying medicine at Kiev University. After 18 months in Nikolskoye, he went on to specialize in venereology in Kiev. Shortly thereafter, he gave up a career in medicine for writing. All his life he was

sceptical to the Soviet system and used his satire against the regime. He worked on his main work, *The Master and Margarita*, from 1928 until his death. The novel was not published in his lifetime.



Figure 50. Mikhail Afanasyevich Bulgakov

(1891–1940), rosyjski lekarz i pisarz, którego historie są oparte na jego doświadczenie jako lekarz wiejski w małej wiosce o nazwie Nikolskoje w prowincji Smolensk. Nikolskoje było jego pierwszym miejscem pracy po studiach medycznych na Uniwersytecie w Kijowie. Po 18 miesiącach w Nikolskoje, udał się na specjalizację z wenerologii w Kijowie. Wkrótce potem zrezygnował z kariery medycznej. Został pisarzem. Całe życie był sceptyczny do systemu sowieckiego i jego satyra skierowana była przeciwko reżimowi. Pracował nad swoją główną pracą *Mistrz i Małgorzata*, od 1928 aż do śmierci. Powieść nie została opublikowana za jego życia.

CUCKOOPINT SIGN

Purging, cold clammy skin, with swelling of the tongue. Indicates poisoning from arum maculatum. Also known as Arum Maculatum sign.



Figure 51. Cuckoopint sign

OBJAW CUCKOOPINT

Biegunka oraz zimna, wilgotna skóra, z obrzękiem języka. Wskazuje na zatrucie *Arum maculatum* (Obrazkiem Plamistym). Objaw również znany jako OBJAW „ARUM MACULATUM”.

CULLEN SIGN

In the skin surrounding the navel appear pale blue staining. These are secondary changes in the peritoneum following a hematoma in acute pancreatitis.



Figure 52. Cullen sign

OBJAW CULLENA

W obrębie skóry otaczającej pępek występują bladoniebieskie przebarwienia. Są to wtórne zmiany w następstwie krwiaka otrzewnej w ostrym zapaleniu trzustki.

THOMAS STEPHEN CULLEN

(1869-1953), was a Canadian gynecologist associated with Johns Hopkins Hospital. Cullen was educated at the Toronto Collegiate Institute and the University of Toronto, graduating from the latter school with a Bachelor of Medicine degree in 1890. He began studying at Johns Hopkins University the next year, before traveling to Germany and studying at Johannes Orth's laboratory at the University of Göttingen in 1893. From 1893 to 1896, Cullen was in charge of gynecological pathology at Johns Hopkins, and in 1919 he was named a professor of clinical gynecology. Cullen researched gynecological diseases including uterine cancer and ectopic.

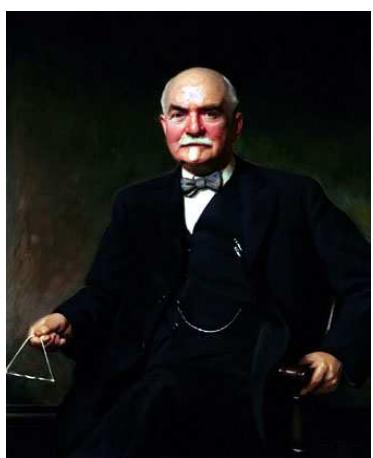


Figure 53. Thomas Stephen Cullen

(1869-1953), był kanadyjskim ginekologiem związанныm z Johns Hopkins Hospital. Kształcił się w Toronto Collegiate Institute i University of Toronto, uzyskując tytuł Bachelor of Medicine w 1890 roku. Rozpoczął

studia na Uniwersytecie Johns Hopkinsa w kolejnym roku, wyjeżdża do Niemiec i studiuje w Johannes Orth laboratorium na Uniwersytecie w Getyndze w 1893 roku. Od 1893 do 1896 roku, Cullen był odpowiedzialny za oddział ginekologicznej patologii w Johns Hopkins, a w 1919 roku został mianowany profesorem ginekologii klinicznej. Cullen badał schorzenia ginekologiczne, w tym raka macicy i ciążę pozamaciczną.

ACKNOWLEDGEMENT:

Figure 14, 42

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